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CERVICAL METASTASES FROM SQUAMOUS CELL CARCINOMA OF THE LARYNX.*†

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Cancer of the larynx has been recognized as a lethal disease since ancient times. Although attacked by constantly improving surgical methods for more than a century, as well as by radiologic methods for half that time, this disease continues to be the cause of many deaths. Since the pioneering efforts of such men as Garcia, Buck, Billroth, Mackenzie, Jackson, and Coutard, impressive, and indeed extraordinary, successes have been attained in the eradication of primary malignant tumors in the larynx.[21,48] On the other hand, efforts to eliminate metastatic laryngeal cancer have met with more limited success, and today, most authors ascribe the majority of failures in therapy of laryngeal cancer to failure in eliminating such metastatic growths.[12,16,22,25]

Squamous cell carcinomas of the larynx, accounting for at least 95 per cent of all instances of laryngeal cancer,[21] may conceivably metastasize by inhalation, by contact, and

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by arterial, venous and lymphatic channels, although instances of spread by all but the latter two routes are medical rarities.[47,53] Venous metastatic growths, although not uncommonly found at necropsy, are quite infrequent during most of the course of this disease.[7,21,40,43] Many such instances result from the invasion of adjacent veins by tumor cells in cervical lymph nodes.[53] The lymphatic channels are the predominant routes of metastasis from carcinoma of the larynx.[53] Since the work of Delamere, Poirier and Cunéo, and of Rouvière, it has been known that nearly all of the lymphatic drainage from the larynx and adjacent structures passes to lymph nodes in the cervical region, predominantly those of the deep cervical or jugular chains. Although a few extracervical pathways exist, clinical and pathologic findings indicate that infraclavicular metastases from carcinoma of the larynx are quite uncommon.[7,26,38,40,43]

Stated differently, most of the fatalities in this disease, due to metastatic spread, may be attributed to such spread in the cervical region.[3] Surgical removal of the regional cervical lymph nodes was suggested as early as 1904 by de Santi and 1906 by Crile, but it was largely replaced by roentgen therapy after about 1920. More recently, renewed interest exists in radical surgical attack on the cervical areas of metastatic spread, as evidenced by the work of such men as del Sel and Agra, Ogura,[33] Schall and Bocca. Some controversy exists concerning the criteria and indications for such therapy, for it has been well known since the work of Krishaber,[23,24] and of Isambert, in the 1870's, that carcinomas of the larynx behave in widely differing manners, including their likelihood to metastasize. Careful statistical studies of large groups of cases should, as in the cases of lip and cervical cancers, provide valuable information about this likelihood of spread.[47] Few such studies have been made, in detail, about metastases from laryngeal carcinoma, especially regarding the influence on the probability of nodal spread of various factors present at the time of diagnosis, and thus on which principles of therapy can be based.

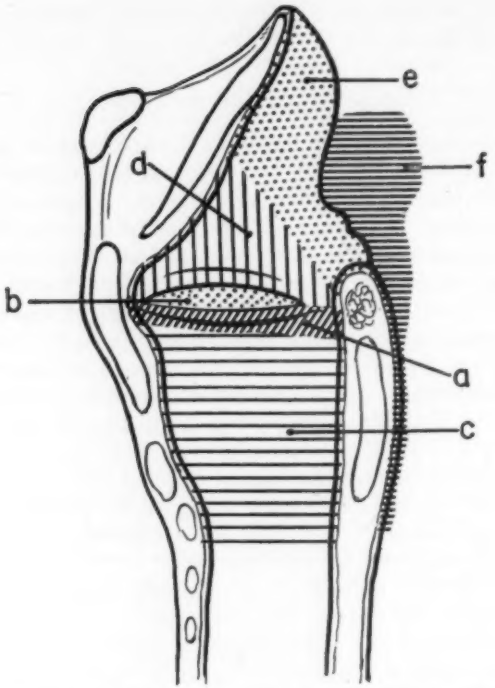
The purpose of this study was to determine from a large series of cases what influence various factors had on the

incidence of cervical metastasis from squamous cell carcinoma of the larynx.

MATERIALS AND METHODS.

From all the cases of squamous cell carcinoma of the larynx seen at the Mayo Clinic in the five-year period 1945 through 1949, 602 cases were selected for study. The criteria for selection were: *1.* That histologic proof of the diagnosis could be made from available materials; *2.* that the diagnosis was first made in the period noted. The clinical records of these cases were reviewed in detail, and permanent sections, prepared from the laryngeal lesions and in some cases from cervical lymph nodes, were examined. The tumors were graded according to the method introduced and subsequently modified by Broders,[8,9,11] denoting the degree of cellular anaplasia exhibited microscopically. In addition, each tumor was classified as infiltrative or as *in situ*, according to whether or not malignant cells had invaded the nonepithelial underlying tissues.[1,10,51] These classifications, regarding the histologic grade and the infiltrative or *in situ* nature of the tumors, were made from tissues obtained at the time of diagnosis. Gross specimens of 254 surgically removed larynges were also examined. The subsequent courses of the patients were then studied from the records of follow-up examinations and from information obtained from a follow-up letter survey and other communications. The anatomic limits[21,29,49] of the larynx were defined as the cricotracheal junction inferiorly, the free edge of the epiglottis superiorly, and the lateral walls of the hypopharynx laterally (see Fig. 1).

The matter of classifying the tumors as to sites of origin and existing locations and extent presented great difficulty. The number of methods described for classifying laryngeal tumors is perhaps an indication of the failure of any to be very satisfactory. Our method for this study was: *1.* Each tumor was carefully described in anatomic terms only; *2.* 21 categories, incorporating various factors of location, size, extent and stage, were delineated, and the cases were placed in the appropriate categories; *3.* these categories were combined to form nine groups, and then to form two groups.



Anatomic areas of the larynx used in classifying laryngeal cancer. a. Cordal area; b. Ventricular area; c. Subglottic area; d. Vestibular area; e. Marginal area; f. Pyriform sinuses and postcricoid area.

The combinations were made after the results were known, the different categories being combined in a manner that simplified, but did not distort, the results.[25] Additionally, the presence or absence of gross extension of tumor outside the defined limits of the larynx at the time of treatment was noted.

The figure represents, on an outline drawing of the larynx, the anatomic areas delineated, which are those used by Pietrantoni. The definitions of the nine groups, in terms of which most of the results are presented, and the combinations of these into two groups, are given in Table I. Tumors in

Group I were confined to the cordal area entirely, with or without fixation, or extended outside this area only to the ventricle. Group II included the cordal tumors which extended into the subglottic or the vestibular area but not into both. Group III included the cordal tumors which extended into both the subglottic and vestibular areas, but not into other areas. Group IV tumors were the ventricular tumors.

TABLE I.

METHOD OF CLASSIFICATION OF CARCINOMA OF THE LARYNX ACCORDING TO SITE.

Origin	Group	Definition
	Cordal Tumors	
Cordal area	I	Confined to cordal area, or with extension to ventricle only.
Cordal area	II	Extension to subglottic or vestibular area but not to both.
Cordal area	III	Extension to subglottic and vestibular areas but not to other areas.
	Noncordal and diffuse tumors	
Ventricular area	IV	All ventricular tumors.
Subglottic area	V	All subglottic tumors.
Vestibular area	VI	All vestibular tumors.
Marginal area	VII	All marginal tumors.
Pyriform and postcricoid areas	VIII	All pyriform sinus and postcricoid tumors.
Uncertain or cordal area	IX	Diffuse tumors, and cordal tumors extending beyond limits of Groups I, II and III.

Group V tumors were the subglottic tumors. Group VI tumors were the vestibular tumors. Group VII tumors were the marginal tumors. Group VIII included the tumors located in the pyriform sinuses and postcricoid area. Group IX included the tumors of probable cordal origin that extended beyond the limits of tumors of Groups I, II, and III, and also widespread or diffuse tumors of uncertain origin. The more numerous categories delineated primarily in this study, which included two or more groups for each of the nine just noted, were found to add little to the results, and so are omitted.[25] Considered as cordal tumors were all those included in Groups I, II and III, while considered as noncordal and diffuse tumors were those included in the remaining groups.

The criteria selected for denoting cervical metastases evolved from a philosophy akin to that expressed by Blair and co-workers, namely, that only the passage of time disproves the presence of metastases. A single clinical estimate of the presence or absence of metastases is known to be grossly erroneous,[6,33,37] while insistence on histologic proof of metastases makes most series so highly selective that serious question of the validity of the sample as representative of the whole exists; also, errors of omission as high as 33 per cent may occur from reliance on histologic proof alone.[42]

We have chosen a middle ground and have considered as having a metastasis any case with: *1.* Histologic evidence of a cervical metastasis; or *2.* historical evidence of a cervical metastasis, meaning evidence, from multiple follow-up examinations and information from other sources, of the evolution of an enlarging hard cervical mass which, with little doubt, was a cervical metastasis. Because single clinical opinions or doubtful information was not considered adequate to fulfill this criterion, and because the minimal follow-up period was five years, adequate knowledge of the subsequent courses of patients, both with and without cervical masses at the time of treatment, was available. In effect, time was substituted for histologic slides, in many instances, and done so, we feel, without serious danger of error. Those cases in which a so-called "local recurrence" of the laryngeal growth occurred prior to the later appearance of a cervical metastasis were excluded from the results, because the assumption that such a metastasis was present at the time of original treatment is open to serious question. Such an assumption is quite reasonable if a metastasis occurs before such a "recurrence," or if no "recurrence" occurs. Considered as adequately followed were: *1.* Those cases with cervical metastases, as just defined; *2.* those cases without such metastases which were followed for a minimum of five years. Ninety-seven patients had died before the presence or absence of metastases could be ascertained by our criteria. Of the remaining 505 cases, 402 (79.6 per cent) were adequately followed.

RESULTS.

The results are presented in three sections: those in the first section, general findings, were derived from the total series of 602 cases; those in the second section, non-infiltrative or *in situ* tumors, were derived from the 56 cases of this nature, except for the findings regarding metastases. These findings and those of the third section, metastases from infiltrative tumors, were derived from the 402 adequately followed cases. After excluding the eight cases in which "local recurrences" of the tumors occurred before the cervical metastases were discovered, 394 cases remained. Forty of these had non-infiltrative tumors, and 354 had infiltrative tumors.

A. General Findings; Age and Sex Incidence—The average age of the patients in this series was 58.0 years, with a range from 18 to 83 years. Sixty-eight per cent of the patients were in the sixth and seventh decades, and 86 per cent were in the fifth, sixth and seventh decades, while only 3 per cent were less than 40 years of age. There were 550 men and 52 women, a ratio of 10.6 to 1. The female patients were slightly younger than the male patients by an average of 2.8 years.

Histologic Grade—Eight and one-tenth per cent of the patients had tumors Graded 1; 56.4 per cent had tumors Graded 2; 30.4 per cent had tumors Graded 3; and 5.1 per cent had tumors Graded 4. The great majority (86.8 per cent) had tumors Graded 2 or 3. Tumors of the higher two grades included 34.3 per cent of the tumors in men, compared to 48.0 per cent of the tumors in women.

Site Incidence—Sixty-six per cent of the total were classified as cordal tumors, including Groups I, II, and III (see Table II). Cordal tumors confined to the cordal area, or with extension to the ventricle only, included 42.0 per cent of the total (Group I), while 16.9 per cent were cordal tumors with extension to the subglottic area or the vestibule (Group II), and 7.1 per cent were cordal tumors with extension to both the subglottic area and the vestibule (Group III). The remainder (34.0 per cent of the total) were classified as

noncordal and diffuse tumors, including Groups IV through IX. Ventricular tumors (Group IV) included only 0.3 per cent of the total, and subglottic tumors (Group V) only 1.7 per cent. Vestibular tumors (Group VI) included 12.8 per cent of the total, while 5.1 per cent were marginal tumors (Group VII), 8.6 per cent were pyriform sinus and postcricoid tumors (Group VIII), and 5.5 per cent were diffuse tumors, or widespread tumors of cordal origin (Group IX) (see Table II). The only significant difference in site incidence found between the sexes was in the pyriform sinus

TABLE II.

SITE INCIDENCE OF CARCINOMA OF THE LARYNX: 602 CASES.

Group	Cases	
	Number	Per cent
Cordal tumors	(397)	(66.0)
I	252	42.0
II	102	16.9
III	43	7.1
Noncordal and diffuse tumors	(205)	(34.0)
IV	2	0.3
V	10	1.7
VI	77	12.8
VII	31	5.1
VIII	52	8.6
IX	33	5.5
Total	602	100.0

and postcricoid tumors (Group VIII), which included 5.8 per cent of the tumors in men but 38.6 per cent of the tumors in women, emphasizing the well-known female predilection for tumors in this area.

Correlations—Several analyses were made to ascertain whether any correlations existed between the various general factors studied. The average ages of patients with tumors graded the same were compared and were found to increase gradually as the grade became higher. The maximal variation between patients with tumors Graded 1 and those with tumors Graded 4, was 5.0 years. No sex difference was found. The average ages of patients whose tumors were in similar sites were compared, and no significant differences were found,

indicating that no relation existed between the patient's age and the location of the tumor. The average duration of symptoms before diagnosis was found to be significantly shorter in patients whose tumors were Graded 3 or 4 than in patients whose tumors were Graded 1 or 2, while the average duration of symptoms before diagnosis was found to be significantly longer in patients with cordal tumors than in patients with tumors located elsewhere in the larynx. Seventy-seven and eight-tenths per cent of the cordal tumors were Graded 1 or 2, while only 38.5 per cent of the noncordal and diffuse tumors were Graded 1 or 2, and the farther from the vocal cords a tumor was located, the greater was the likelihood of its being Graded 3 or 4 (see Table 3). Thus,

TABLE III.

HISTOLOGIC GRADE OF CARCINOMA OF THE LARYNX IN DIFFERENT SITES: 602 CASES.

Histologic grade	Group. I	II	III	IV	V	VI	VII	VIII	IX	Cordal	Noncordal and Diffuse	Total Cases	Total Per cent
Total Cases	252	102	43	2	10	77	31	52	33	397	205	602	
	Per cent	Per cent	Per cent	Per cent	Per cent	Per cent	Per cent	Per cent	Per cent	Per cent	Per cent	Cases	Per cent
1	15.5	4.9	0	0	0	5.2	0	0	3.0	11.1	2.4	49	8.1
2	69.4	59 9	67.5	100.0	50.0	37.7	45.2	19.2	42.5	66.7	36.1	339	56.4
3	14.7	32.3	32.5	0	50.0	49.3	51.6	55.8	39.4	21.2	48.3	183	30.4
4	0.4	2.9	0	0	0	7.8	3.2	25.0	15.1	1.0	13.2	31	5.1
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	602	100.0

80.8 per cent of the pyriform sinus and postcricoid tumors were Graded 3 or 4, compared to only 15.1 per cent of the tumors confined to the cordal area.

B. Noninfiltrative or In Situ Tumors—There were 56 patients with tumors classified as *in situ*. The age and sex incidence of these patients and the durations of their symptoms were found not to differ from those of the total series. In contrast to the tumors of the total series, 55 per cent of these tumors were Graded 1, 96 per cent were Graded 1 or 2, and none were Graded 4. In addition, 95 per cent of these tumors were cordal tumors (Groups I, II and III), and 89 per cent were confined to the cordal area and ventricle (Group

I). No cervical metastases occurred among the 40 cases of non-infiltrative tumors which met the criteria for follow-up, but in 11 of these patients "local recurrences" developed after treatment of the laryngeal growths. The "recurrent" growths were infiltrative tumors in seven of the 11 instances.

C. Metastases From Infiltrative Tumors—As discussed earlier, the findings concerning metastases from infiltrative tumors were derived from the 354 cases which met the criteria for follow-up and which did not have "local recurrences" prior to the discovery of metastases. The over-all incidence of metastasis from these cases was 31.4 per cent.

TABLE IV.

INCIDENCE OF CERVICAL METASTASIS FROM CARCINOMA OF THE LARYNX IN DIFFERENT SITES: 354 CASES.

Group	Cases	Per cent with metastasis
Cordal tumors	(230)	(17.4)
I	138	1.4
I—Confined to cordal area	(121)	(0.8)
I—To ventricle	(17)	(5.9)
II	59	32.2
II—To subglottis	(33)	(30.3)
II—To vestibule	(26)	(34.6)
III	33	57.6
Noncordal and diffuse tumors	(124)	(57.3)
IV	0	0
V	5	60.0
VI	53	43.4
VII	14	78.6
VIII	29	75.9
IX	23	52.2
Total	354	31.4

Relation to Site of Tumor—The incidence of cervical metastasis from cordal tumors (Group I, II and III) was 17.4 per cent, while that from noncordal and diffuse tumors (Groups IV through IX) was 57.3 per cent (see Table 4). Among the cordal tumors, 57.6 per cent of those involving both the subglottis and the vestibule (Group III) had metastases, 32.2 per cent of those involving either the subglottis or the vestibule but not both (Group II) had metastases, and only 1.4 per cent of those confined to the cordal area and ventricle (Group I) had metastases. Among the tumors of Group

II, 30.3 per cent of those involving only the subglottis in addition to the cordal area had metastases, and 34.6 per cent of those involving only the vestibule in addition to the cordal area had metastases. Among the tumors of Group I, only one of 121 tumors confined to the cordal area had a metastasis (0.8 per cent), this being a tumor involving both cords with fixation. Only one of 17 tumors with extension to the ventricle had a metastasis. No ventricular tumors (Group IV) had metastasis, and of the five subglottic tumors (Group V), three had metastases. Forty-three and four-tenths per cent of the vestibular tumors (Group VI) had metastases, 78.6 per cent of the 14 marginal tumors (Group VII) had metas-

TABLE V.

INCIDENCE OF CERVICAL METASTASIS FROM CARCINOMA OF THE LARYNX OF DIFFERENT HISTOLOGIC GRADES: 354 CASES.

Histologic grade	Cases	Per cent with metastasis
1	12	0.0
2	207	20.8
3	115	47.0
4	20	70.0
Total	354	31.4

tases, 75.9 per cent of the pyriform sinus and postcricoid tumors (Group VIII) had metastases, and 52.2 per cent of the diffuse tumors (Group IX) had metastases. A large variation in the incidence of metastasis from tumors in different locations was evident, this incidence rising rapidly as the distance of the tumor from the vocal cords increased.

Relation to Grade—No cervical metastases occurred from tumors Graded 1. Among the tumors Graded 2, 20.8 per cent had metastases, among the tumors Graded 3, 47.0 per cent had metastases and among the tumors Graded 4, 70.0 per cent had metastases (see Table V). A large variation in the incidence of metastasis from tumors of different grades was evident, the likelihood of metastasis rising rapidly with increasing grade.

Relation to Sex—Although the small number of women (31) studied and the previously noted differences in the

sites of tumors between the two sexes make comparison dubious, the incidence of cervical metastasis from tumors in men was 29.7 per cent, compared to 48.0 per cent from tumors in women.

Relation to Duration of Symptoms and to Age—The incidence of cervical metastasis was found to increase slightly as the duration of symptoms became longer, but not significantly. Similarly, this incidence was found to be slightly greater among older patients than among younger patients, but again not significantly.

Correlations—Several analyses were made to determine whether further correlations existed between the incidence of metastasis and the several factors studied. No significant relation was found between the duration of symptoms and the incidence of metastasis when cases with tumors in similar sites were compared. This further emphasized that the presence of symptoms for a long time did not, alone, significantly increase the likelihood of metastasis. Likewise, no significant relation was found between the age of the patient and the incidence of metastasis when cases with tumors in similar sites were compared. This further emphasized that age, alone, did not significantly alter the likelihood of metastasis.

Although the incidence of metastasis was higher among cases with tumors in certain sites, it was also noted that tumors in these sites were generally of higher grade. Because higher-grade tumors in general were noted to metastasize more frequently than lower-grade tumors, the question arises as to which factor, site or grade, was the more significant determinant of the likelihood of metastasis. Considering tumors located in similar sites, the incidences of metastasis found from tumors of different grades were compared (see Table VI). No metastases occurred from tumors Graded 1. Only two of the tumors confined to the cordal area and ventricle (Group I) had metastases, but both were Graded 3, although only 17 per cent of these 138 tumors were graded above 2. The incidence of metastasis was higher from tumors Graded 3 than from tumors Graded 2 among

all other cordal tumors (Groups II and III), and among all cordal tumors together (Groups I, II and III). Since only two cordal tumors were Graded 4, they are not considered. A similar but more marked increase in the incidence of metastasis as the grade becomes higher was found among the noncordal and diffuse tumors (Groups IV through IX), rising from 44.4 per cent from tumors Graded 2, to 62.7 per cent from tumors Graded 3, and to 77.8 per cent from tumors Graded 4. The relation between incidence of metastasis and grade of tumor was thus found to exist independently of the site of the tumor.

TABLE VI.

INCIDENCE OF CERVICAL METASTASIS FROM CARCINOMA OF THE LARYNX OF DIFFERENT HISTOLOGIC GRADES AND IN DIFFERENT SITES: 354 CASES.

	Group						
	I	II and III	IV, V, VI and IX	VII and VIII	Cordal	Noncordal and diffuse	Total
Histologic grade	Cases 138	92	81	43	230	124	354
	Per cent with metastasis						
1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
2	0.0	41.1	31.4	90.0	14.2	44.4	20.8
3	9.1	44.1	57.1	70.8	30.4	62.7	47.0
4	0.0	0.0	77.8	77.8	0.0	77.8	70.0
Total	1.4	41.3	46.9	76.7	17.4	57.3	31.4

From the same data, and considering only tumors of the same grade, the incidences of metastasis from tumors of different sites were compared (see Table VI). These incidences were found to be greater as the distance of the tumors from the cordal area increased, independently of the grade of the tumors. For example, metastases occurred from 14.2 per cent of the cordal tumors Graded 2, but from 44.4 per cent of the noncordal and diffuse tumors Graded 2; from 30.4 per cent of the cordal tumors Graded 3, but from 62.7 per cent of the noncordal and diffuse tumors Graded 3. These comparisons indicate that both the grade and the site of the tumors had correlations with the likelihood of metastasis, and that these correlations exist independently of each other.

Relation to Gross Extension Outside the Larynx—A comparison was made between the incidences of metastasis from tumors extending outside the anatomic limits of the larynx as determined at the time of treatment, and from tumors without such extension. Excluding cordal tumors confined to the cordal and ventricular areas, none of which had such gross extension, by definition, it was found that the incidence of metastasis increased from 47.4 per cent for tumors without extension to 57.8 per cent from tumors with extension.

D. Accuracy of Clinical Estimation—A clinical impression of the presence of cervical metastasis, made at the time of diagnosis, was found to be incorrect once in every 12.6 such estimates, and a clinical impression of the absence of metastasis at that time was found to be incorrect once in every 5.5 such estimates. Over all, any estimate made was incorrect in 16.4 per cent of the cases.

COMMENT.

Discussion may be confined to comment about three subjects: *1.* Criticisms of the methods used; *2.* comparison of the results with those of others; *3.* interpretation of the results.

Criticisms of the Methods—Important criticisms of the methods used in this study are felt to arise from two factors: the proportion of adequately followed patients and the criteria used for defining a metastasis. Because a five-year minimal, and hence a ten-year maximal, follow-up period was considered necessary, many patients were first seen seven to ten years ago. Information concerning some of these patients, no longer troubled by their tumors, or dead from other causes than the tumors, was not obtained, and they were not, therefore, adequately followed. The proportion of those who were adequately followed, 79.6 per cent, is comparable to that of most series of cases reported. Unfortunately, some patients died from their tumors, or from other causes before the presence or absence of metastases could be determined, and these patients had to be excluded. The exclusion of these patients from the results dealing with metastases may have

affected the results to some degree. If the results are in error from this account, the reported incidences of metastasis are probably too low. The rationale used in defining a metastasis was discussed earlier. In summary, the criteria were selected to make this study as complete as possible, so that the results might be considered applicable to all patients with squamous cell carcinoma of the larynx who appear for care, and at the same time to avoid the errors of using only histologic evidence, or only brief clinical evidence of metastases.[25] The only studies of large series of cases of this kind which present detailed information about cervical metastases, those of Pietrantoni and of Ogura,[34] were based on histologic evidence of metastasis, from selected surgical cases. Comparisons of their findings with those of this study, noted below, may also be thought of as comparing the different methods used.

Comparison of the Results with Those of Others—The age and sex incidence in this series does not differ from that usually reported for squamous cell carcinoma of the larynx, [21,27,31,32,49] nor does the distribution of the tumors according to grade,[32,49] although the latter has seldom been reported. Most reports [21,27,49,50] agree with the finding in this report that cordal tumors occur more frequently than noncordal tumors, although a few authors[29,31] found the reverse to occur. Other reports[15,21,28] comparing the grades of these tumors to their sites, have also pointed out that cordal tumors are of considerably lower grade, generally, than are tumors arising elsewhere in the larynx. The relative frequency of pyriform sinus and postcricoid tumors in women found in this study has been often noted by others.[46] Significant differences between the general findings in this series and those in other reports were not found.

The findings in this series regarding cases with non-infiltrating or *in situ* tumors are strikingly similar to those reported by Altmann, Ginsberg and Stout. Their conclusions—that such tumors rarely occur outside the cordal area, that they occur in patients with the same age and sex incidence and similar durations of symptoms as patients with laryngeal cancer in general, that they comprise approximately 9

per cent of all laryngeal malignant lesions, and that they do not metastasize—are substantiated. Other reports[30,51] do not differ significantly from these.

Comparisons of the incidences of metastasis found in this study, with those reported by most authors, are seriously hampered by confusions and differences in classifications, and because most authors report only surgically treated cases, these often after considerable selection of cases. It is felt that selection of cases on the basis of "operability," which implies also the state of general health, although useful in reporting results from operations, may fail to give accurate information about the disease in general. In spite of these difficulties, general comparisons can be made. The over-all incidence of cervical metastasis in patients undergoing laryngofissure or laryngectomy has been reported as 13.5 per cent to 23 per cent.[13,15,17,22] Among patients undergoing laryngectomy only, this incidence has been reported as 23 per cent and 30 per cent.[5,14,16] Orton stated that metastases develop in 30 per cent of all cases of laryngeal cancer. We feel that the incidence found in this study, 31.4 per cent, represents the incidence to be expected from a series including all patients presenting themselves for the first time. The findings in this study relating the incidence of metastasis to the site of laryngeal tumors are in good agreement with the general opinions and findings of others,[26,29,49] namely, that squamous cell carcinoma confined to the cordal area rarely metastasizes, and that those tumors located in such areas as the pyriform sinus and postcricoid region metastasize frequently, in fact usually. More specific comparisons with other studies were not feasible, except with the few studies reported in detail. Those of Pietrantoni and of Ogura,[34] noted earlier, are examples of such comparable studies. Pietrantoni reported a series of 466 patients who underwent radical neck dissection and laryngectomy, and Ogura reported a series of 59 patients who underwent operation. Both studies were based on histologic evidence of metastasis in the excised specimens. In spite of considerable differences in the selection and classification of cases, the findings in our study show excellent agreement with the findings of these authors

regarding the incidences of metastasis found for tumors in different sites.[25] No reports were found describing the relation between the incidence of metastasis and the age and sex of the patients, the duration of their symptoms, or the grade of the tumor. The higher incidence of metastasis found from noncordal tumors, compared to that from cordal tumors, has been considered by others[12,21,26,52] to be due to the higher grades of such tumors, to the more abundant lymphatic networks present in these areas, or to both. The findings in this study indicate that both the grade and the site, independently, are related to the greater likelihood of metastasis from these tumors. The somewhat increased likelihood of metastases found from tumors extending outside the larynx, compared to those without such extension, is in agreement with Ogura's findings[34] but is less marked. The considerable inaccuracy of clinical estimation of the presence or absence of cervical metastases found in this study certainly is in accord with the many similar reports of others.

Interpretation of Results—The findings in this study are felt to be primarily confirmatory. As was intimated earlier, surgical treatment of carcinoma of the larynx has tended to become more radical in recent years, primarily because of increasing recognition of the role played by cervical metastases in causing failures; however, recognition of the general failure of radiation therapy to control such metastases, and the ability to perform more radical neck operations without prohibitive mortality and morbidity, have also encouraged this trend.[2] In spite of this predilection for more radical surgical attack, little detailed information is available about the likelihood of cervical metastases and the factors related to that likelihood. This kind of information should be available, if these operations are to be followed by consistent results, and is necessary to formulate logical indications for such procedures.

The findings in this report indicate that laryngeal cancer in certain locations so frequently metastasizes to the cervical lymph nodes that such areas of spread should be removed at the time of the primary treatment of the laryngeal growth. Impalpability of suspected cervical metastases does not alter

this viewpoint. The findings in this study also indicate that the histologic grade of the tumor should be included with the site of the tumor as an important factor affecting the likelihood of cervical metastasis, and that the age and sex of the patient and the duration of his symptoms should not be included. In addition, the findings provide further evidence that *in situ* carcinomas of the larynx do not metastasize, and that clinical impressions of whether cervical metastases are present are inaccurate.

SUMMARY AND CONCLUSIONS.

A study was made of 602 consecutive and histologically verified cases of squamous cell carcinoma of the larynx seen at the Mayo Clinic during a five-year period. General findings regarding age and sex of patients, duration of symptoms, histologic grade and nature of tumors, and site and extent of tumors in this series were similar to these findings in most other reports. The occurrence of cervical metastases was studied in 394 of these cases that met certain criteria. No cervical metastases occurred in cases with non-infiltrative or *in situ* tumors. Cervical metastases occurred in 31.4 per cent of cases with infiltrative tumors. Such metastases occurred rarely from tumors confined to the cordal area, but occurred with increasing frequency the farther tumors were located from this area. Metastases occurred in about one-third of cases with cordal tumors that extended to the vestibule or the subglottis and of cases with vestibular tumors. More than half of the cases with cordal tumors extending to both the subglottis and the vestibule, cases with diffuse tumors and cases with subglottic tumors had cervical metastases. Metastases occurred in about three-fourths of the cases with marginal tumors and of cases with tumors in the pyriform sinuses and postcricoid area. No metastases occurred from tumors Graded 1, but metastases occurred from tumors Graded 2 or more, with rapidly increasing frequency as the grade advanced. The occurrence of metastases was unrelated to the patient's age, and the duration of his symptoms was probably unrelated to the patient's sex and was somewhat more likely in patients with tumors that extended out-

side the larynx than in patients with tumors that did not extend outside the larynx. Metastases were found to be more frequent from tumors of higher than of lower grade, regardless of their location, and were also found to be more frequent the farther the tumors were located from the cordal area, regardless of the grade of the tumor. Predictions about cervical metastases based on palpation of the neck at the time of diagnosis were frequently inaccurate.

In conclusion, the findings of this study indicate that among various items of information available at the time of treatment, the site of the tumor, the histologic grade of the tumor, and the microscopic infiltrative nature of the tumor, each significantly affect the likelihood of occurrence of cervical metastases from patients with squamous cell carcinoma of the larynx. These factors, therefore, should be important determinants of the indications for radical procedures designed to remove cervical areas of metastatic spread in this disease.

REFERENCES.

1. Altmann, Franz, Ginsberg, Irving and Stout, A. P.: Intraepithelial Carcinoma (Cancer In Situ) of the Larynx. *A.M.A. Arch. Otolaryngol.*, 56:121-133, Aug., 1952.

2. Beahrs, O. H., Devine, K. D. and Pemberton, A. H.: Combined Operations for Intraoral and Laryngeal Carcinoma. *A.M.A. Arch. Surg.*, 67:215-226, Aug., 1953.

3. Beahrs, O. H. and Jordan, G. L., Jr.: Bilateral Radical Cervical Dissection for Malignant Lesions of the Head and Neck. *Proc. Staff Meet., Mayo Clin.*, 27:449-454, Nov. 5, 1952.

4. Blair, V. P., Brown, J. B. and Byars, L. T.: Our Responsibility Toward Oral Cancer. *Ann. Surg.*, 106:568-576, Oct., 1937.

5. Bocca, Ettore: Functional Problems Connected With Bilateral Radical Neck Dissection. *Jour. Laryngol. and Otol.*, 67:567-577, Sept., 1953.

6. Brachetto-Brian, D. and Samengo, L. A.: Las Adenopatias Metastasicas Cervicales no Palpables en el Cancer de la Laringe Confirmadas por el Vaciamiento Profilactico. *Prensa Med. Argent.*, 37:3033-3034, Dec. 15, 1950.

7. Braund, R. R. and Martin, H. E.: Distant Metasasis in Cancer of the Upper Respiratory and Alimentary Tracts. *Surg., Gynec. and Obstet.*, 73:63-71, July, 1941.

8. Broders, A. C.: Squamous-cell Epithelioma of the Lip: A Study of 537 Cases. *J.A.M.A.*, 74:656-664, Mar. 6, 1920.

9. Broders, A. C.: Cancer's Selfcontrol. *Med. Jour. and Rec.*, 121:133-135, Feb. 4, 1925.

10. Broders, A. C.: Carcinoma In Situ Contrasted With Benign Penetrating Epithelium. *J.A.M.A.*, 99:1670-1674, Nov. 12, 1932.

11. Broders, A. C.: The Microscopic Grading of Cancer. *Surg. Clin. N. Amer.*, pp. 947-962, Aug., 1941.

12. Carroll, W. W. and Shields, T. W.: In-continuity Neck Dissection for Laryngo-hypopharyngeal Carcinoma. *Quart. Bull. N. W. Univ. Med. School*, 28:46-54, Spring, 1954.

13. Clerf, L. H.: Cancer of the Larynx; An Analysis of 250 Operative Cases. *Arch. Otolaryngol.*, 32:484-498, Sept., 1940.

14. Clerf, L. H.: Discussion. Proceedings of the Second National Cancer Conference, New York, Amer. Cancer Soc., Inc., Vol. 1, pp. 462-463, 1952.

15. Clerf, L. H., Putney, F. J. and O'Keffe, J. J.: Carcinoma of the Larynx. The Laryngoscope, 58:632-641, July, 1948.

16. Cody, C. C., Jr.: Treatment of Carcinoma of the Larynx; a Statistical Study of Results. The Laryngoscope, 59:621-638, June, 1949.

17. Colledge, Lionel and Peacock, Roydon: An Analysis of 126 Cases of Malignant Disease of the Upper Air Passages Treated During a Period of Ten Years, 1921-1930. *Jour. Laryngol. and Otol.*, 47:161-187, March, 1932.

18. Crile, George: Excision of Cancer of the Head and Neck, with Special Reference to the Plan of Dissection Based on 132 Operations. *J.A.M.A.*, 47:1780-1786, Dec. 1, 1906.

19. Delamere, G., Poirier, P. and Cunéo, B.: "The Lymphatics," Translated by C. H. Leaf, 301 pp. Constable & Company, Ltd., London, 1913.

20. Isambert: Contribution a l'Etude du Cancer Laryngé. *Ann. d. mal. de l'Oreille, du Larynx.* 2:1-23, 1876.

21. Jackson, Chevalier and Jackson, C. L.: "Cancer of the Larynx," 309 pp. W. B. Saunders Co., Phila., 1939.

22. Jackson, C. L., Blady, J. V., Norris, C. M. and Maloney, W. H.: Cancer of the Larynx; Five Year End Results in a Series of Patients Treated Between 1930 and 1942. *J.A.M.A.*, 138:1080-1083, Dec. 11, 1948.

23. Krishaber, M.: Contribution a l'Etude du Cancer du Larynx. *Gaz. Hebd. de Med.*, 16:518-523, Aug. 15, 1879.

24. Krishaber, M.: Quoted by Leborgne, F. E.: Classification of Carcinoma of the Larynx. The Laryngoscope, 63:1089-1095, Nov., 1953.

25. Kuhn, A. J.: Cervical Metastases From Squamous Cell Carcinoma of the Larynx; a Clinico-pathological Study. *Thesis, Grad. School, Univ. Minn.*, 1956.

26. Lewis, R. S.: Discussion on the Management of Cervical Metastases From Laryngeal Carcinoma. *Proc. Roy. Soc. Med.*, 46:481-484, July, 1953.

27. MacKenty, J. E.: Cancer of the Larynx. *Arch. Otolaryngol.*, 3:205-232, March; 305-337, April, 1926.

28. Martin, Hayes: Cancer of the Head and Neck. III—Cancer of the Larynx. *J.A.M.A.*, 137:1366-1369, Aug. 14, 1948.

29. Martin, H. E.: Selection of Treatment for Cancer of the Larynx. *Ann. Otol., Rhinol. and Laryngol.*, 49:728-735, Sept., 1940.

30. Miller, A. H. and Fisher, H. R.: Carcinoma-in-situ of the Larynx. *Ann. Otol., Rhinol. and Laryngol.*, 62:358-370, June, 1953.

31. Negus, V. E.: Intrinsic Cancer of the Larynx; Review of a Series of Cases. *Proc. Roy. Soc. Med.*, 40:515-522, July, 1947.

32. New, G. B., Figi, F. A., Havens, F. Z. and Erich, J. B.: Carcinoma of the Larynx; Methods and Results of Treatment. *Surg., Gynec. and Obstet.*, 85:623-629, Nov., 1947.

33. Ogura, J. H.: Laryngectomy and Radical Neck Dissection for Carcinoma of the Larynx. *Trans. Am. Acad. Ophthal.*, 55:786-806, Nov.-Dec., 1951.

34. Ogura, J. H.: Surgical Pathology of Cancer of the Larynx. The Laryngoscope, 65:867-926, Oct., 1955.

35. O'Keefe, J. J.: Secondary Radical Neck Dissection. *Trans. Am. Laryngol., Rhinol. and Otol. Soc.*, pp. 40-45, 1955.

36. Orton, H. B.: Evaluation of Radical Neck Dissection. *Trans. Am. Acad. Ophthal.*, 58:30-34, Jan.-Feb., 1954.

37. Pietrantoni, Luigi: Il Problema Chirurgico delle Mestastasi Linfoghiandolari Cervicali del Cancro della Laringe. *Arch. Ital. Otol.*, Suppl. 14, 64:1-37, 1953.

38. Price, L. W.: Metastasis in Squamous Carcinoma. *Am. Jour. Cancer*, 22:1-16, Sept., 1934.

39. Rouviere, Henri: "Anatomy of the Human Lymphatic System," 318 pp., Translated by M. J. Tobias. Edwards Brothers, Inc., Ann Arbor, Mich., 1938.

40. Rubenfeld, Sidney and Kaplan, Gustave: Carcinoma of the Larynx with Distant Lympatic Metastases. *A.M.A. Arch. Otolaryngol.*, 56:255-261, Sept., 1952.

41. de Santi, P. R. W.: The Lymphatics of the Larynx and Their Relation to Malignant Disease of That Organ. *Lancet*, 1:1710-1713, June 18, 1904.

42. Saphir, Otto and Ambomin, G. D.: Obscure Axillary Lymph-node Metastasis in Carcinoma of the Breast. *Cancer*, 1:238-241, July, 1948.

43. Sawyer, K. C., Papayannopoulos, G. J. and McClure, H. E.: Large Bowel Obstruction Caused by Metastatic Carcinoma Originating in the Larynx; Report of a Case and Review of the Literature. *A.M.A. Arch. Surg.*, 68:126-130, Jan., 1954.

44. Schall, L. A.: Evaluation of Total Laryngectomy. *Trans. Am. Acad. Ophthal.*, 58:26-29, Jan.-Feb., 1954.

45. del Sel, Jaime and Agra, Alejandro: Cancer of the Larynx: Laryngectomy with Systematic Extirpation of the Connective Tissue and Cervical Lymph Nodes as a Routine Procedure (Laryngeal Cancer and Lymphatic Metastasis). *Trans. Am. Acad. Ophthal.*, 51:653-654, July-Aug., 1947.

46. Semon, Felix: Some Points in the Diagnosis and Treatment of Laryngeal Cancer. *Brit. Med. Jour.*, 1:241-248, Feb. 2, 1907.

47. Taylor, G. W. and Nathanson, I. T.: "Lymph Node Metastases," 498 pp.; preface; p. 6. Oxford Univ. Press, New York, 1942.

48. Thomson, StClair: The History of Cancer of the Larynx. *Jour. Laryngol. and Otol.*, 54:61-87, Feb., 1939.

49. Thomson, StClair and Colledge, Lionel: "Cancer of the Larynx," 244 pp. Kegan Paul, Trench, Trubner & Co., Ltd., London, 1930.

50. TUCKER, GABRIEL: Observations on the Inter-relationship, Diagnosis and Treatment of Benign and Malignant Tumors of the Larynx (With Special Reference to the Prevention and Cure of Laryngeal Cancer). *Trans. Am. Laryngol., Rhinol. and Otol. Soc.*, pp. 29-33, June, 1941.

51. VOTAW, R.: Carcinoma *In Situ* in Otolaryngology. THE LARYNGOSCOPE, 62:973-997, Sept., 1952.

52. WALSH, T. E.: The Classification of Carcinoma of the Larynx. THE LARYNGOSCOPE, 57:414-418, June, 1947.

53. WILLIS, R. A.: "The Spread of Tumours in the Human Body," 447 pp. Butterworth & Co., Ltd., London, 1952.

POSTGRADUATE CONFERENCE IN OTOLARYNGOLOGY, UNIVERSITY OF MICHIGAN MEDICAL SCHOOL.

The Department of Postgraduate Medicine at the University of Michigan Medical School announces the Otolaryngology Conference to be held at the University Hospital, Ann Arbor, Michigan, on April 18, 19 and 20, 1957, under the direction of Dr. A. C. Furstenberg, Chairman of the Department of Otolaryngology at the University of Michigan Medical School.

GUEST LECTURERS.

Dr. Percy Ireland, Toronto, Ontario; Dr. Richard Bellucci, New York City; Dr. Theodore E. Walsh, St. Louis, Missouri; Dr. Howard House, Los Angeles, California; and Dr. Charles E. Kinney, Cleveland, Ohio.

MEMBERS OF STAFF.

Dr. A. C. Furstenberg, Dr. J. H. Maxwell, and Dr. J. E. Magielski.

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ATTICOMASTOIDECTOMY.*†

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The title I have chosen, namely atticomastoidectomy, has several synonyms, some of which are modified radical mastoidectomy, Bondy mastoidectomy and antro-adital-atticotomy.

In our clinic, this procedure is used for patients who have a postero-superior *marginal* or *attic* perforation with persistent discharge of months', or years', duration which has failed to clear up in response to frequent local treatments of the ear over a period of six weeks. In my opinion the presence of cholesteatoma is nearly always a definite indication for this operation. Symptoms of extension of infection to the labyrinth, facial nerve or dura call for immediate operative treatment. I think an acute exacerbation of a chronic infection or the presence of a complication is not always a contraindication to use of the modified radical mastoidectomy.

The well-known purposes of the operation are (1) to eradicate a chronic focus of infection which is a potential threat to life, (2) to obtain a dry ear, and (3) to maintain or improve the hearing to a useful level, that is 30 db or better at the 500, 1000, and 2000 frequencies. The value of this operation has been well described by Juers,[1,2,3] Baron,[4,5,6] Thorburn,[7] Moore,[8] Dingley,[9] Zwiefach[10] and many others. My chief excuses for bringing this operation to your attention are to present additional evidence of its merits in properly selected cases, and to refute some of the doubtful statements made in recent papers.

Some of these quotations which our experience contradicts are as follows: "If the ossicular chain is interrupted, hearing is likely to be better if the tympanic membrane is re-

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moved;" "forty per cent of patients having a modified operation will eventually require a complete radical operation;" "useful hearing following the modified operation depends upon maintaining the integrity of the ossicular chain;" "it is best to leave a dislocated incus;" "a modified operation is never indicated when complications are present."

During the three-year period from June 1, 1953, to June 1, 1956, my associates and I have operated on 122 chronically discharging middle ears, having attic or postero-superior marginal perforations. Seventy-eight per cent had cholesteatoma. The atticomastoidectomy procedure was used for 45, and the complete radical operation was used for 77. Fifty-four per cent of the chronically discharging middle ears seen during the three-year period had *non* marginal perforations and did not require operative treatment. The average healing time after atticomastoidectomy was eight weeks, and all except two ears were dry in three months. These two patients later required a revision and complete radical mastoidectomy. Skin grafts were not used.

The healing time in our cases is at least as short as that reported by most of those who do utilize skin grafts. In my opinion the reasons for the rapid healing in most of our cases have been frequent careful mechanical cleansing, avoidance of ear drops and *especially the fact* that the patient is instructed to use boric acid powder in a powder blower at home twice daily. There are at least two reasons to explain the observed efficacy of boric acid powder in postoperative cavities. The desirability of maintaining an acid reaction on the skin has been established by many observers. I believe that boric acid is just enough of a protoplasmic poison to discourage formation of granulations without impairing the regrowth of epithelium.

The indications for surgery when there is a marginal or attic perforation may be listed as, *1.* Persistent discharge, even though small in amount, for months' or years' duration which has failed to clear up despite adequate local treatment during a period of six weeks; *2.* Presence of pseudocholesteatoma (the term advocated by Day[11]); *3.* Symptoms

of extension of the infection to the labyrinth, facial nerve or dura; *4*. X-ray evidence of a large cholesteatoma.

Some of the operative methods we find useful are: general anesthesia; surital induction and maintenance with nitrous oxide plus Trilene and oxygen through an endo-tracheal tube; the use of an endaural incision, a Jordan-Day bone engine, head light and loupe magnification, and electro-coagulation to stop bleeding. We determine the type of operation by the extent of the disease found at the time of operation. I agree with Baron[5] and Juers[2] that it shortens the healing time and that it is perfectly safe to save the matrix on the medial wall of the epitympanum and aditus. I follow the advice of Juers[3] and Baron[4] in cutting the flaps to suit the anatomy and operative defect. The best post-operative hearing occurred when the operator either intentionally or unintentionally performed what Juers has described as myringostapediopexy. Fifty-four per cent of the ears from which the incus and head of the malleus were removed had hearing at or above the 30 db level. The good hearing in these cases was due to the presence of a mobile membrane providing sound pressure transformation for the oval window and sound protection for the round window, as described by Wullstein.[12] When doing an atticomastoidectomy we aim to produce what Wullstein[12] describes as his Type 3 tympanoplasty. Fig. 1 (after McGuckin[13]) shows the anatomical result desired.

A study of the case histories of these patients has led me to record what might be called guiding principles: *1*. X-ray failed to diagnose cholesteatoma in 55 per cent of the cases; *2*. X-ray examination seldom tells you when to operate; *3*. Middle ear effusion frequently coexists with attic cholesteatoma (do myringotomy and aspiration of middle ear fluid at the time of operation; *4*. Do not overlook allergic factors; *5*. Recurrent acute otitis media is not chronic otitis media; *6*. Gram stain *smears* and sensitivity cultures can be very important in selection of the most effective antibiotic for use during the post-operative period; *7*. Many ear drops are harmful (contact dermatitis or allergic sensitization); *8*. In three cases boric acid powder stopped the discharge after

Neo-cortef had failed; *9.* Save as much drum as possible; *10.* Let the type of operation depend upon the extent of disease found at operation; *11.* Much hearing loss can be prevented by operating when the cholesteatoma is small; *12.* Clear up any coexisting sinus infection; *13.* Remove the adenoid if it is enlarged.

As is well-known, most serious complications are the result of an acute exacerbation of chronic middle ear and

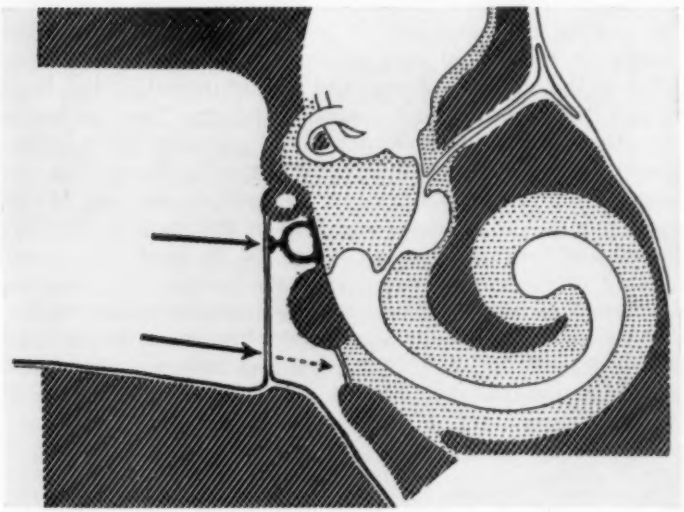

Fig. 1.

mastoid infection. All too often the patient, and even sometimes the doctor, is complacent about the existence of a small amount of discharge from an attic or postero-superior marginal perforation. In my opinion the infection in all such ears should be eradicated by atticomastoidectomy. Unwillingness to consider operative treatment must be based either upon ignorance of the danger of sudden flare-up of a chronic infection or fear that the operation will seriously impair the hearing. In this series of 45 cases, 66 per cent of the

cars with good cochlear function had post-operative hearing at or above the 30 db level, and in only one instance was the post-operative hearing below the 30 db level if it had been that good pre-operatively. The importance of using the modified operation when possible is obvious, if one recalls that following a complete radical the hearing usually drops to the 55 or 60 db level. For some years I have avoided destroying the annulus and made no attempt to close the Eustachian tube when doing a complete radical. In 10 of 77 radical cases a drum, furnishing sound protection for the round window, has regenerated and the hearing in all 10 ears returned to the 20 to 30 db level.

Our experience is very much in line with that of Dingley and Zwiefach,[10] who say that best hearing is usually present in cases having a wide aditus and no incus. In most of their cases they removed the incus if it was present, and also usually removed the head of the malleus. In our experience the ears having poor post-operative hearing usually had a retracted poorly mobile *pars tensa* and a blocked Eustachian tube secondary to nasal allergy, or to recurrent sinus infection. Better control of these factors and better patient cooperation will, I believe, increase the percentage of ears with good post-operative hearing.

Post-operative care is just as important as the operation and should, if possible, be carried out with magnification by the surgeon doing operation, or someone equally experienced. I remove the packing on the second or third day, because it is well known that moisture is harmful to squamous epithelium. I use gentle spot suction to remove clots and fibrin from the ear canal only, and follow this with insufflation of boric acid powder. The patient leaves the hospital on the fifth or sixth post-operative day with instructions to use boric acid powder in a hand powder blower at home daily until the ear is dry. We have return visits twice a week the first two weeks and thereafter once a week until healing is complete, usually by the end of the eighth week. If the drum is retracted and moves poorly, I inflate, do myringotomy and aspirate the mucus or serum in the middle ear. This may need to be repeated. The patient is

instructed to return for cleansing of the cavity about every six months, or oftener if there is any discharge. We have many post-operative cavities which require cleansing only once a year.

A wet ear after the normal two-months' healing time is usually due to one of the following causes: *1.* Contact dermatitis due to antibiotic or sulfonamid drops; *2.* Persistent granulations; *3.* Allergic dermatitis; *4.* Infrequent cleansing.

At the time of operation one is always faced with the question, will the modified procedure eradicate the disease and produce a dry ear? If the incus is embedded in granulation tissue it is not possible to say whether the long process is necrotic, so I think it is better to remove it if the hearing is below the 25 db level. I think there is a better chance of a columella effect if the incus and head of the malleus are removed. If the incus is fixed, dislocated, or is carious it should be removed.

Nearly all of the patients who had the incus removed had hearing between the 20 and 30 db levels in the speech frequencies, providing they had good bone conduction, a movable membrane protecting the round window, and a patent Eustachian tube. The principles to follow in planning this type of operation have been well outlined by Zollner[14] and by Kobrak.[15]

Table I, indicating whether or not the incus was present, shows what degree of post-operative hearing may be expected, and suggests that if both windows are functioning normally loss of the incus need not be responsible for a hearing loss of more than 20 db below normal threshold. The Table includes only the ears which had good cochlear function.

The following case reports illustrate the value of atticomastoidectomy in properly selected cases:

W. C.—male, age 42. C.C. dizziness of two weeks' duration. Intermittent discharge from the right ear for a period of eight years. Right ear: there was pus and cholesteatomatous debris in an attic perforation. The postero-inferior part of the drum moved freely. A right atticomastoidectomy was performed on July 13, 1954. There was a cholesteatoma sac external to the incus. The incus was not disturbed, and the matrix covering the incus was allowed to remain. Pre-operative hearing was at

TABLE I.

PATIENTS WITH ATTIC CHOLESTEATOMA OR POSTERO-SUPERIOR MARGINAL PERFORATIONS TREATED BY ATTICOMASTOIDECTOMY.

Age	Incus	Cholesteatoma	Post Op. Cavity Dry In	Hearing—DB Loss At 500 1000 2000 Pre-Op.	Post-Op.
41	Removed	Yes	7 weeks	20-15-15	30-35-35
49	Absent	Yes	7 weeks	55-55-80	15-20-60
21	Absent	Yes	7 weeks	25-25-25	20-20-15
51	Removed	No	8 weeks	30-30-20	50-45-45 Large dry perforation
20	Removed	No	7 weeks	20-20-25	30-25-35
10	Not Rem.	Yes	8 weeks	20-20-05	10-15-10
42	Absent	Yes	8 weeks	50-50-50	20-20-20
16	Not Rem.	Yes	5 weeks	20-15-15	10-10-15
20	Removed	Yes	9 weeks	45-40-30	25-30-30
18	Not Rem.	No	7 weeks	25-15-5	20-20-30
18	Not Rem.	No	7 weeks	25-25-10	15-15-10
21	Removed	Yes	8 weeks	10-10-10	10-10-15
21	Removed	No	8 weeks	45-30-30	20-20-25
37	Removed	No	8 weeks	55-55-45	30-30-25
31	Not Rem.	No	13 weeks	10- 5 -10	15-15-10
42	Not Rem.	No	6 weeks	15-15-15	30-25-15
6	Removed	No	6 weeks	35-30-45	25-20-25
6	Not Rem.	No	6 weeks	20-15-25	30-20-25
27	Removed	No	7 weeks	20-30-30	30-30-30
21	Removed	No	6 weeks	10-10-20	20-20-20
39	Not Rem.	Yes	6 weeks	20-15-10	20-15-10
13	Removed	No	5 weeks	40-40-30	30-25-15
19	Removed	No	3 months	5-15-15	20-15-10
42	Removed	Yes	6 weeks	60-60-50	40-40-40
8	Removed	No	8 weeks	30-40-35	20-45-35
23	Removed	Yes	5 weeks	25-35-25	20-20-20
34	Absent	Yes	6 weeks	50-55-45	40-50-45
8	Removed	No	6 weeks	30-30-45	25-25-50
33	Absent	Yes	7 weeks	25-15-15	35-25-30
50	Absent	Yes	8 weeks	40-40-30	50-50-40

the 15 db level in speech range; post-operative hearing was at the 20 db level throughout the speech range. The mastoid cavity was healed in six weeks and has remained dry up to the present time.

R. S.—male, age 27. C.C. intermittent discharge from the right ear since age of three years. The drum was markedly retracted and moved poorly. There was a postero-superior marginal perforation containing granulation tissue. X-ray examination showed a radiolucent area in the region of the right mastoid antrum. A small amount of foul discharge continued for two months despite local treatment. Suction with

the pneumatic otoscope always brought mucopus out of the postero-superior marginal perforation. A right atticomastoidectomy was performed on Aug. 17, 1954. The incus was embedded in a mass of granulations and was removed. Pre-operative hearing was at the 30 db level throughout the speech range; the post-operative hearing remained at the 30 db level. Mastoid cavity healed in seven weeks. The *pars tensa* has good mobility, as shown by the use of the pneumatic otoscope.

G. V.—male, age 10 years. C.C. intermittent discharge from the right ear since infancy. The right ear canal contained thick yellow mucopus. There was a postero-superior marginal perforation containing cholesteatomatous debris. Treatment: right endaural atticomastoidectomy. The antrum contained a large cholesteatoma sac 15 mm. in diameter. The incus and malleus were not removed but were covered with a flap from the superior canal wall. The cavity was dry in eight weeks. Pre-operative hearing 20-20-15; post-operative hearing 10-15-10. The *pars tensa* moves freely.

M. N.—female, age eight years. C.C. discharge from the left ear since age of six weeks. For two weeks prior to admission had recurrent nausea, vomiting and vertigo. Left ear canal contained mucopus and there was a polyp in the postero-superior marginal perforation. Treatment: Adenoidectomy and atticomastoidectomy. There was granulation tissue and a small amount of pus in the left epitympanum, aditus and antrum. The incus and head of the malleus were removed. Pre-operative hearing 30-30-45; post-operative hearing 25-25-50. The cavity was dry in six weeks; no recurrence of vertigo.

C. W.—male, age 22 years. C.C. intermittent discharge from the right ear since early childhood. Examination showed mucopus in the right ear canal. There was a large polyp filling the inner end of the canal. Treatment: Endaural atticomastoidectomy. A cholesteatoma sac filled the epitympanum, aditus and antrum. The disease had caused resorption of th incus and head of the malleus. The cholesteatoma matrix was allowed to remain on the medial wall of the epitympanum Pre-operative hearing 30-40-35; post-operative hearing 20-20-15, because the *pars tensa* moves freely and there is an inflatable tympanic cavity protecting the round window. Healing was complete in five weeks.

W. S.—male, age 42 years. C.C. discharge from the right ear since 10 years of age, increasing in amount the past 30 days. Had pain in the right ear and unsteadiness when walking the week prior to admission. Chills and fever for 48 hours. Physical examination: The right ear canal was filled with mucopus, and there was a polyp against the drum; temp. 103°; WBC 12,200; culture hemolytic Staph coagulase positive sensitive to penicillin. Diagnosis: Acute exacerbation of chronic mastoiditis and septicemia. Treatment: Fortified procaine penicillin two million units IMq. four hours, and right endaural atticomastoidectomy. The antrum, which had been enlarged by the cholesteatoma sac, contained thick yellow pus under pressure. Resorption of bone had exposed the dura and the lateral sinus, and there was a perisinus abcess. The incus had been destroyed. The head of the malleus was removed. Pre-operative hearing 50-45-50; post-operative hearing 20-20-20, because the *pars tensa* moved freely. The cavity was healed in eight weeks.

SUMMARY AND CONCLUSIONS.

1. Atticomastoidectomy was used for 45 ears having chronic attic and mastoid disease.

2. There were no deaths and no facial paralyses.

3. The average healing time was eight weeks.

4. Sixty-six per cent had post-operative hearing at or above the 30 db level in the speech range.

5. In only one instance was the hearing below the 30 db level if it had been that good before operation.

6. In 12 ears with the incus destroyed by infection or removed at operation the hearing in the speech range remained at or above the 30 db level.

7. Useful hearing is chiefly dependent upon the presence of an intact movable *par tensa*, a patent Eustachian tube and absence of scar tissue around the oval and round windows.

8. Skin grafts are not necessary to produce rapid healing.

9. Preservation of cholesteatoma matrix in the epitympanum can hasten healing and help preserve useful hearing.

10. There would be fewer unfortunate delays in advising operation if we stressed the fact that 46 per cent of the patients having chronically discharging middle ears actually have chronic attic and mastoid disease requiring surgical treatment.

REFERENCES.

1. Juers, A. L.: Current Concepts of Attic and Middle Ear Disease. *J.A.M.A.* 160:451-455, Feb. 11, 1956.

2. Juers, A. L.: Modified Radical Mastoidectomy. *A.M.A. Arch. Otol.*, 57:245-256, March, 1953.

3. Juers, A. L.: Preservation of Hearing in Surgery for Chronic Disease. The Laryngoscope, 64:235-251, April, 1954.

4. Baron, S. H.: Modified Radical Mastoidectomy. *A.M.A. Arch. Otol.*, 49:280-302, March, 1949.

5. Baron, S. H.: Conservation of Hearing in the Modified Radical Mastoidectomy. *Trans. Am. Laryngol., Rhinol. and Otol.*, 209-245, 1954.

6. Baron, S. H.: Preservation of Hearing in Mastoid Surgery; Technical Considerations. *Trans. Am. Acad. Opth. and Otol.*, 59:385-396, May-June, 1955.

7. Thorburn, I. B.: Factors That Influence the Hearing Result in Surgery of Chronic Otitis Media. *Jour. Laryngol. and Otol.*, 68:739-757, Nov., 1954.

8. Moore, J. A.: Endaural Modified Radical Mastoidectomy. *Trans. Am. Laryngol., Rhinol. and Otol.*, 44-57, 1953.

9. Dingley, A. R.: Some Limitations of Radical Mastoidectomy and the Value of Early Conservative Surgery. *Jour. Laryngol. and Otol.*, 69:361-373, June, 1955.

10. Dingley, A. R. and Zwiefach, E.: Some Observations on Attic Disease and Its Treatment. *Jour. Laryngol. and Otol.*, 68:127-139, March, 1954.

11. Day, K. M.: Primary Pseudocholesteatoma of the Ear. *A.M.A. Arch. Otol.*, 34:1144-1151, Dec., 1941.

12. Wullstein, H.: Theory and Practice of Tympanoplasty. *Trans. Am. Laryngol., Rhinol. and Otol.*, 548-565, 1956.

13. McGuckin, F.: In Discussion of Zollner. *Jour. Lar. and Otol.*, 69:687-690, Oct., 1955.

14. Zollner, F.: The Principles of Plastic Surgery of the Sound-Conducting Apparatus. *Jour. Laryngol. and Otol.*, 69:637-652, Oct., 1955.

15. Kobrak, H. G.: Recent Advances in Diagnosis and Treatment of Middle Ear Deafness. *Trans. Am. Laryngol., Rhinol. and Otol.*, 162-172, 1956.

INTERNATIONAL VOICE CONFERENCE.

The International Voice Conference will be held in Chicago May 20-22, 1957. Distinguished Laryngologists, Physiologists, Physicists, and Voice Scientists from outstanding research centers in United States, Europe and Asia, will appear on the program.

The various days will be devoted to the following subjects: Monday—Research on Physiology of Voice Production; Tuesday—Clinical Procedures in Diagnosis and Training; Wednesday—Relation of Hearing to Voice.

Participation in the Conference will be by prior registration.

Further information may be obtained from Dr. Hans von Leden, 30 North Michigan Ave., Chicago 2, Illinois.

THE EFFECT OF THYROIDECTOMY ON THE NASAL MUCOSA OF EXPERIMENTAL ANIMALS.*†

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(By Invitation).

Since myxedema was first described by Curling[1] in 1850 voluminous literature has appeared concerning the clinical and biochemical aspects of this example of hormonal deficiency. Of interest to the otorhinolaryngologist are the symptoms of deafness (conductive and perceptive), nasal obstruction and dysphonia, which may reputedly result from a decrease in the level of circulating thyroxin. The experiments about to be described were designed to make possible the actual study of microscopic changes in the nasal mucous membrane produced by deficiency of thyroid activity.

HISTORICAL REVIEW.

The clinical otolaryngological significance of hypothyroidism has been excellently described by a number of observers. In a series of 65 cases analyzed by Bryant[2] 23 had symptoms referable to the ear, 18 to the nose and 13 to the throat; while 11 had miscellaneouus symptoms such as "choking spells" and pain referable to the Vth cranial nerve. This author stated further: "A problem confronting every otolaryngologist is that of nasal obstruction, watery discharge and repeated colds which persist even after apparently well-indicated operations have been performed and consideration given to other causes, such as allergy and dietary indiscretions." Forman[3] posed the suggestion that vasomotor rhinitis,

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even when of atopic origin, became manifest only in the presence of provoking or aggrevating factors; and he listed thyroid deficiency as a frequent trigger mechanism.

McLaurin[4] stated: "Because of the negative and unimpressive characteristics of most of the signs and symptoms of hypometabolism, the condition frequently escapes detection; therefore, before an obscure disease is placed in the waste basket of borderline allergy or neurosis one may well investigate the metabolic status of the patient." Proetz,[5] in a classic study, related his experience with a group of 130 patients, 66 of whom were first suspected of hypothyroidism on the singular observation of the nasal mucosa. From these cases stemmed his remark that the membrane changes of myxedema were of two types: the first was red, dry and chapped or irritated, while the second was pale, wet and boggy. He noted that headache and nasal obstruction were prominent features of hypothyroidism, and most characteristic was the utter failure of both nose and patient to respond to the usual measures of therapy.

In 126 out of 202 patients seen by Hollender[6] and complaining of otolaryngic difficulty had minus BMR readings, and of these 60 per cent responded to thyroid therapy. He concluded that a lack of appreciation of the part played by endocrine dysfunction, more especially hypometabolism, in certain ear, nose and throat disorders could logically explain the futility of some time honored local procedures. The acceptance of a low basal metabolism rate as a criterion for mild hypothyroidism was condemned by Walsh,[7] who advocated the administration of small doses of thyroid extract for any case of vasomotor rhinitis which exhibited lethargy in addition to the usual complaints. Chavanne,[8] in an ingenious experiment in 1937, measured the nasal secretions following thyroidectomy.

MATERIALS AND TECHNIQUE.

The dog was selected as the experimental animal because of its ability to withstand surgical procedures well; moreover, the animal husbandry department at this activity had excellent care and quarters available for the animals. All

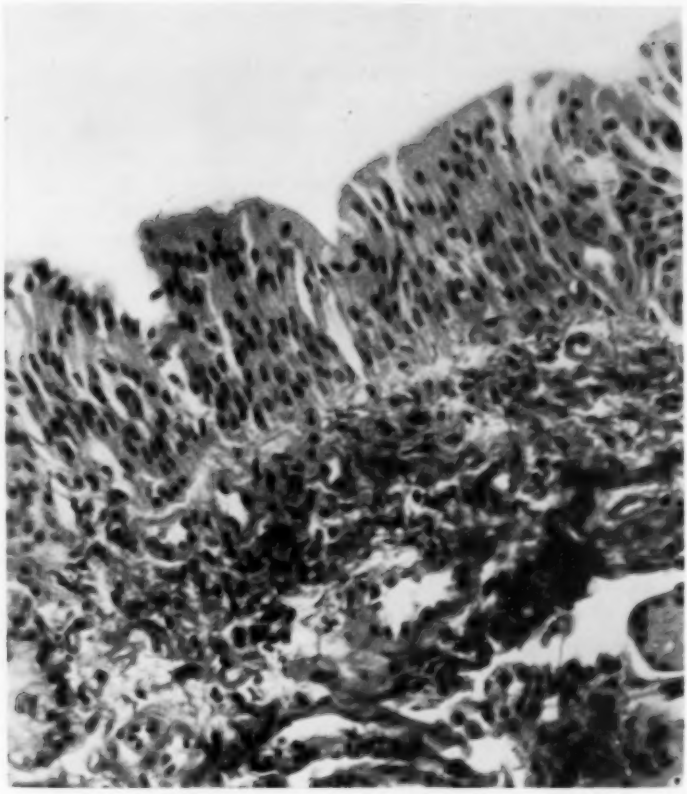

Fig. 1—Control.

were maintained on a vitamin-rich, high caloric diet to exclude dietary deficiency as a possible variable. The dogs were divided into two groups with two animals in Group I and eight dogs in Group II. In Group I an attempt was made to produce a state of hypothyroidism by partial thyroidectomy realizing that rapid regeneration from unresected tissue might make this impossible. The second group was placed in the athyroid condition by total thyroidectomy. Pre-

thyroidectomy nasal mucosal biopsies served as controls for both groups.

Under intravenous sodium pentothal anesthesia each animal underwent initial biopsy of the mucosa of the right lateral nasal wall. Varying amounts of thyroid tissue were excised from the dogs in Group I, and the animals were then maintained on Hytakerol mgm., 0.625 t.i.d. and Di-Cal-D caps. 1 b.i.d. to prevent hypoparathyroidism. In Group II complete extirpation of all thyroid gland tissue was carried out, and the four parathyroid glands were dissected free under the operating microscope and implanted in the ribbon muscles. Subsequently serial biopsies were made on the mucosa of the left lateral nasal wall at varying time intervals, and the gross appearance of the membrane was evaluated by means of a nasopharyngoscope from time to time. Four of the animals in Group II were tested for thyroid activity.

RESULTS.

The microscopical findings of the control biopsy specimens were not remarkable. All exhibited the typical pseudostratified ciliated columnar epithelium with normal submucosal layer (see Fig. 1). The two dogs in Group I which were subjected to partial thyroidectomy were sacrificed at three and six weeks respectively, and at autopsy no regrowth of thyroid tissue was noted. The nasal microscopic changes included mucosal thickening and a loss of cilia.

The animals of Group II were sacrificed at the following intervals with the results below.

Time Interval to Biopsy.	Results.
Animal 1—28 days—	No epithelial changes. Oxyphilic degeneration in gland ducts.
Animal 2—40 days—	Metaplasia of epithelium with loss of cilia. Inflammatory infiltration.
Animal 3—40 days—	No demonstrable histological changes noted.
Animal 4—60 days—	Same as Animal 2, plus swelling of the collagen fibers in some areas.

Animals 5, 6, 7 and 8, also of Group II, underwent serial biopsies of the mucosa at 6, 9 and 12-month intervals and were sacrificed one year after thyroidectomy. Radioactive iodin up-take at nine months after total thyroidectomy was

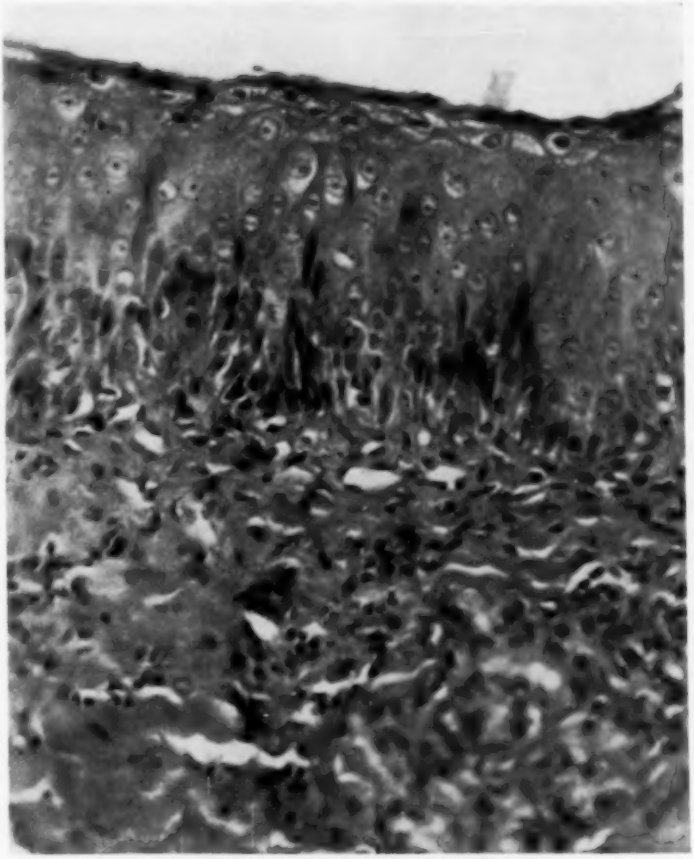

Fig. 2—Squamous metaplasia.

reported as 0 per cent for each dog, and there was an average weight gain of 2.6 kg. for each animal. Inspection of the nasal cavities with a nasopharyngoscope at six and nine months postoperatively revealed a pale, swollen mucosa. At post mortem examinations one year post-thyroidectomy two of the animals revealed similar changes, while in the other two a bluish appearance of the nasal membrane was noted.

There was no histological change noted between the serial antemortem biopsies and the autopsy material, the micropathological features of which were as follows: thickening of the mucosa, predominantly of basal layer with abortive formation of rete pegs, slight squamification, oxyphilic degeneration and/or metaplasis of seromucinous gland ducts, inflammatory infiltration of the acute and chronic types, loss of cilia and swelling of the collagen fibers with fragmentation of fibers in some areas (see Fig. 2).

DISCUSSION.

It is interesting to note the rapidity with which the mucosal changes occurred once thyroid deficiency was induced. It is equally intriguing to speculate as to the mechanism whereby the above micropathological alterations are produced and how they might relate to the clinical phenomena of hypothyroidism in the mucous membranes of man. Obviously the loss of cilia could lead to stagnation of secretions which might, at length, pave the way for secondary infection and edema. This was evidenced in the tendency toward inflammatory infiltration as seen in the slides of the athyroid animals. The increase in the thickness of the epithelium and the tendency to squamification might indicate the reaction to the above inflammatory changes or could conceivably be due to a decreased respiratory rate incident to hypometabolism. Opposite effects are observed in the respiratory membranes of individuals who have undergone laryngectomy.[9] The swelling and separation of the collagen fibers certainly increases the thickness of the lining mucosa and should be a factor in the nasal obstruction observed in hypothyroidism.

Another possible causative factor might be the decrease in blood flow in hypometabolism resulting in poorer nutrition to the mucous membranes; and the vascular stasis could likewise explain the bluish color so frequently observed. In any event, the above microscopic observations explain the dry, irritated chapped appearance described by Proetz. Further experiments are contemplated to determine whether the alterations described in the context are reversible upon the institution of thyroid therapy.

CONCLUSIONS.

Surgically induced thyroid deficiency in the dog produces the following microscopic alterations from normal in the nasal respiratory membrane:

1. Mucosal thickening, especially in the basal layer was apparent.

2. Slight squamification was noted.

3. Oxyphilic degeneration and metaplasia of the seromucinous ducts were present.

4. Acute and chronic inflammatory changes were evident.

5. Cilia were lost.

6. There was swelling of the collagen fibers, and in some areas the fibers were fragmented.

SUMMARY.

Thyroid deficiency was induced in ten dogs by partial and complete thyroidectomy, and the resulting microscopic changes in the nasal mucous membrane were enumerated.

BIBLIOGRAPHY.

1. Cubling, T. B.: Two Cases of Absence of the Thyroid Body, and Symmetrical Swelling of Fat Tissue at the Sides of the Neck, Connected with Defective Cerebral Development. *Med.-Chir. Trans.*, London, 33:303, 1850.

2. Bryant, B. L.: Hypothyroidism in the Practice of Otolaryngology. *Ann. Otol., Rhinol. and Laryngol.*, 45:1060-1068, Dec., 1936.

3. Forman, J.: Hypothyroidism and Vasomotor Rhinitis. *Ann. Otol., Rhinol. and Laryngol.*, 43:279-288, March, 1934.

4. McLaurin, J. W.: Hypometabolism in the Practice of Otolaryngology. *Arch. Otol.*, 41:71-73, Jan., 1945.

5. Proetz, A. W.: The Thyroid and the Nose. *Trans. Amer. Laryngol. Ass'n.*, 1948, and *Ann. Otol., Rhinol. and Laryngol.*, 56:328, June, 1948.

6. Hollender, A. R.: Hypometabolism in Relation to Ear, Nose and Throat Disorders. *Archives of Otolaryngol.*, 63:2, 135-141, Feb., 1956.

7. Walsh, T. E.: Vasomotor Rhinitis. The Laryngoscope, 60:360-367, April, 1950.

8. Chevanne, L.: Nasal Secretion and Endocrine Glands. *Ann. d' Otolaryngol.*, p. 401, May, 1937.

9. Dixon, F. W. and McCall, J. W.: The Nasal Mucosa in the Laryngectomized Patients. *Ann. Otol., Rhinol. and Laryngol.*, 58:22, 535, June, 1949.

UNUSUAL ANATOMICAL VARIATIONS OF THE SPHENOID SINUSES.*

J. C. PEELE, M.D.,

Kinston, N. C.

This paper is based on a study of the anatomical variations of the sphenoid sinuses made in the anatomy laboratory of the University of North Carolina School of Medicine from 1947 through 1956. One hundred and thirty-nine cadavers assigned to medical students were utilized as the source material for this report. Each year at an appropriate time in the student's dissection of the head and neck it was my custom to split the skulls, dissect out and study the sphenoid sinuses, and secure photographs of desired specimens. In this publication emphasis will be placed primarily on variations encountered in this work. A review of the literature on the general subject of the anatomical variations of the sphenoid sinuses is included.

A concept of normal sphenoid sinuses includes two symmetrical cavities with smooth walls and capacity of 5-7 cc., separated by a straight mid-line partition which do not encroach on any of the surrounding structures, and which in turn are not encroached upon by these elements, and each of which contains an ostium of 5 mm. diameter located in the upper fourth of the anterior sphenoidal wall about 5 mm. from the cribriform plate of the ethmoid and mid-line partition of the sphenoid sinuses. I have seen only one specimen that met these requirements. "Normal" sphenoid sinuses are, therefore, quite rare. Variations in the size of the sinuses and thickness of the walls, irregularities of the walls, extension of the sinuses into adjacent structures, or vice versa, are so commonplace that they must be considered as representative of the anatomy of this region.

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The sphenoid sinus has been called the most variable bilateral cavity or organ in the human body. The reasons for these anatomical aberrations are controversial. Pneumatization of the sphenoid bone is a function of the subepithelial layer of the sphenoidal sinus mucosa. If for any reason, such as local infection or malfunction of the endocrine glands, the pneumatizing function of this layer is impaired, then an abnormality of the sinus will develop on one or both sides.

The development of the sphenoid bone is a complicated process. Ten principal centers of ossification arise in the cartilage that corresponds to this bone, one for each great wing, during the eighth week of fetal life (alisphenoid), one for each lesser wing (orbitosphenoid), two for the body between the great wings during the early part of the third month of fetal life (basisphenoid), two for the body between the lesser wings during the second month (presphenoid), and one for each lingula between the alisphenoid and postsphenoid during the fourth month of fetal life. During the first year of postfetal life the several parts of the sphenoid bone coalesce.

There is evidence to show that the bone formed at the line of fusion of bony nuclei is more dense than the bone immediately adjacent to it, and, therefore, it is more resistant to pneumatization. Toldt, Cope, and Congdon utilized this fact to explain the occurrence of partial septa in and the various forms assumed by the sphenoid sinus. Van Gilse, on the other hand, differed from these authors in that he considered that pneumatization of the sphenoid sinus was halted by the presence of softer material such as cartilage, connective tissue, or blood vessels. With these theories explaining the anatomical variations of the sphenoid sinuses as a background specific variations of the sinuses may now be considered.

RUDIMENTARY SINUSES, DISPARITY IN SIZE, AGENESIS.

If for any of the foregoing reasons the development of the sphenoid sinus of either or both sides is retarded or arrested during the late fetal period, or first three years of

postfetal life, then a small pouch-like sinus limited to the area of the cartilaginous nasal capsule will result (see Fig. 1). Occasionally, a specimen is encountered in which the body of the sphenoid bone is solid except for a depression on the anterior wall usually in communication with a large spheno-ethmoidal recess. When the sphenoid sinus is displaced downward and inward by an overriding posterior ethmoidal cell or over-developed sphenoid sinus of the opposite side, it

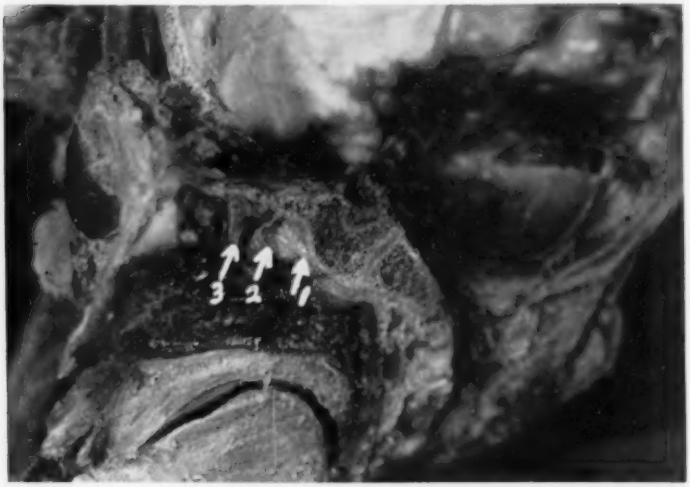

Fig. 1. Pouch-like Rudimentary Right Sphenoid Sinus with Small Ostium—1. Pneumatization Probably Arrested at Birth. Spheno-Ethmoidal Recess—2. Posterior Ethmoid Cell—3.

is likely to be small. When the former is the over-riding cell, the condition may be bilateral.

If pneumatization of the sinus is retarded on one side but unrestricted on the opposite, then the pneumatizing mucosa of the unrestricted side may usurp the bone of the restricted portion during the process of bone absorption. This leads to a marked disparity in size of the two sinuses, as shown in Fig. 2.

Agenesis of the sinuses is reported freely in the literature.

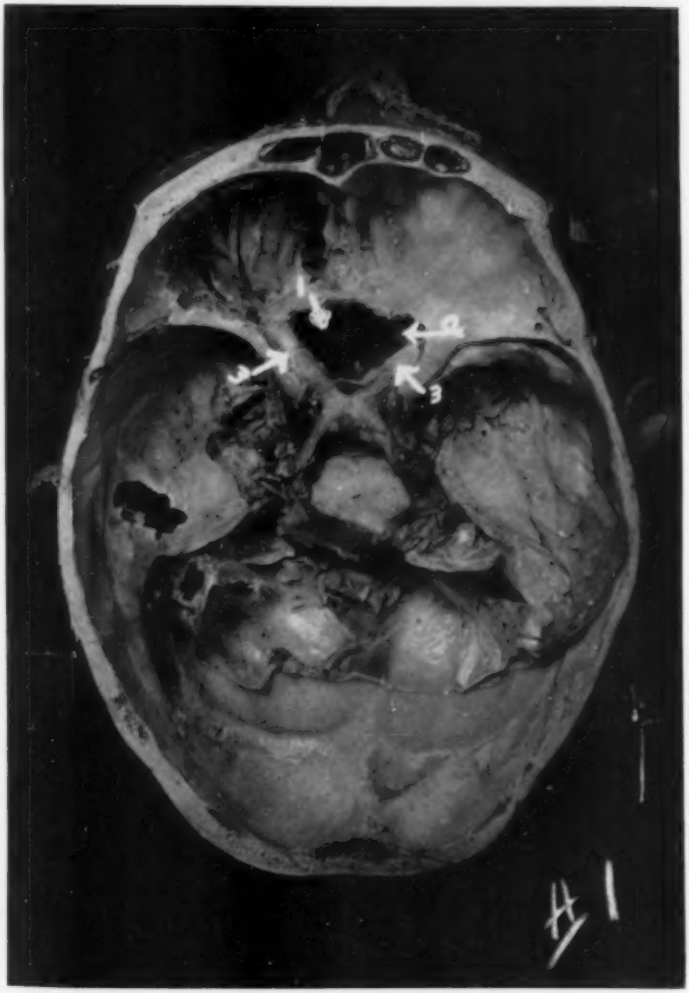

Fig. 2. Disparity in Size Between a Large Left Sphenoid Sinus, dissected—1; and a Small Right Sinus, Unopened—2; the Left Sphenoid Sinus has Pneumatized Both Lesser Wings and is Related to Both Optic Nerves—3.

I did not encounter it, and neither did Douglas, Schaeffer, Canuyt and Terracol, Van Gilse, Van Alyea, and others.

INTER-SINUS SEPTUM.

The median partition of the sphenoid sinuses normally occupies an antero-posterior position and vertical plane, and, due to irregularity in the pneumatization of the sinuses, usually deviates to the right or left posteriorly in the region of the pre- and postsphenoid synchondrosis. The septum may be quite thick or thin depending upon the degree of pneumatization of the sinuses; partly membranous; occupy an oblique, dome-shaped, or almost horizontal plane so that one sinus is superior to the other; and occasionally it may be dehiscent so that the sinuses of opposite sides communicate with one another. Rarely, the septum may be invaded by a posterior ethmoid or supernumerary cell. It has been reported as being absent in specimens in which there appears to be only a single large cavity, but this does not seem logical.

OSTIUM.

The usually round bony sphenoidal sinus ostium is normally located in the upper fourth of the anterior sphenoidal wall several millimeters from the cribriform plate of the ethmoid and inter-sinus septum of the sphenoid, opens into the sphenoethmoidal recess above the level of the highest nasal concha present, and faces anteriorly. The ostia are usually, but not always, located at the same level in the anterior wall.

The ostium may be located near the roof or floor of the sinus and rarely may be found in these areas. More unusual locations have been recorded. It occasionally opens into a posterior ethmoid cell. Onodi described a specimen in which the ostium was located in the posterior part of the hiatus semilunaris, and Dixon found a sphenoid sinus opening into the posterior tip of the superior turbinate. Tunis described an ostium which opened into a canal situated in a bony partition between the posterior ethmoid and sphenoid sinuses.

Van Gilse stated that he had found the sinus aperture in the root of the pterygoid process, and in extreme cases in the orbital process of the palate bone. The conditions are likely to be unilateral.

The bony sinus ostium is characteristically narrowed by the presence of the sphenoidal sinus mucosa, and occasionally may be reduced to an aperture of pin-point or slit-like

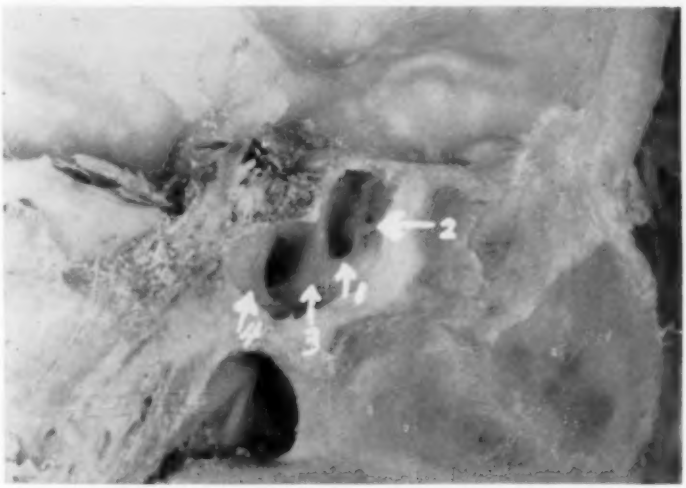

Fig. 3. Left Sphenoid Sinus Extending into the Left Posterior Ethmoid Region at the Usual Site—1; Note Size, Location, and Direction in Which the Sphenoid Sinus Ostium Faces—2; Sphenopalatine Pillar—3; Most Common Site for an Accessory Septum—4.

proportions. A rudimentary sinus may have a small ostium (see Fig. 1), or an ostium so large as to occupy virtually all of the anterior wall without any reduction in size by the sinus mucosa, depending upon the type of rudimentary sinus present. Eadie described an ostium that was fenestrated, presumably with spicules of bone.

The direction in which the ostium faces is influenced by its location in and the slope of the anterior sphenoidal wall, projection of the sinus into the posterior ethmoid or antral

region, or projection of a posterior ethmoid cell into the sphenoidal area, or relations of the sphenoid sinuses of opposite sides to each other, and, therefore, it may point upward, downward, inward, or outward (see Fig. 3).

There is a logical embryological explanation for the single sphenoid sinus with a double ostium that is most rarely encountered, but one should not confuse this extra ostium with a large physiologic dehiscence of the anterior wall transmitting blood vessels, as noted by Van Gilse. The contention of some authors that a completely absent ostium is due to inflamatory closure does not seem tenable. It is possible that an ostium was present in these specimens but overlooked. A double sphenoid sinus of one side each with its own individual ostium has been described, but Van Gilse maintains that a true unilateral double sphenoid sinus cannot exist without a malformation of the nose. It is possible that one of these double sphenoid sinuses so described has been in truth a posterior ethmoid cell.

PROLONGMENTS (RECESSES, BONY DIVERTICULAE) OF THE SPHENOID SINUSES.

Rostrum and Nasal Septum—Extension of the sinus forward in the midline commonly causes a depression in the rostrum of the sphenoid bone, and when this extension is occasionally prolonged downward between the alae of the vomer, an air cell of appreciable proportions develops in the nasal septum.

Ethmoidal Recess—Further laterally the sinus occasionally may invade the posterior ethmoid region. In my experience this is most likely to occur at the postero-lateral-inferior angle of the ethmoidal labyrinth (see Fig. 3). It may extend into the ethmoid region superiorly as far forward as the bulla, thus coming into rather extensive relationship with the orbit, antrum, or supra-orbital extension of the frontal sinus if such be present.

Recess of the Lesser Wing—Laterally and superiorly the sphenoid sinus of the same or opposite side may invade the lesser wing of the sphenoid bone and come into intimate re-

lationship with the optic nerve and ophthalmic artery coursing through the optic canal (see Figs. 2, 15). Complete pneumatization of the anterior clinoid process (see Fig. 4) brings the sinus into additional relationship with the oculomotor and trochlear nerves.

The lesser wing may be invaded by a supra-orbital extension of the frontal sinus or by an anterior ethmoid cell. Also,

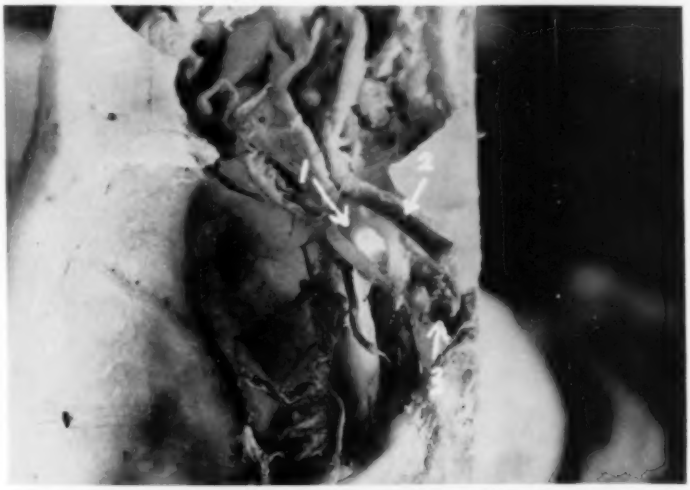

Fig. 4. Left Anterior Clinoid Process Pneumatized by the Left Sphenoid Sinus—1; Optic Nerve—2; Cerebral Portion of the Internal Carotid Artery—3.

a lesser wing may be invaded by an anterior ethmoidal cell, posterior ethmoidal cell, and sphenoid sinus of the same side at the same time. Rarely, a small pneumatic space may exist in the lesser wing independent of any other air cell. This sinus usually drains into a posterior ethmoid cell but may empty into the sphenoethmoidal recess. When a large sinus of the lesser wing exists, it raises the level of the bone above that of the ethmoidal labyrinth so that the bulge may be easily detected in the base of the skull. The abnormal anatomy of this region is extremely complicated. Onodi's

studies established 38 different morphologic relationships bearing on the relations of the optic nerve to the sphenoid sinus and last posterior ethmoid cell in this region.

Palatine (Anterior) Recess—The palatine prolongation places the sphenoid and maxillary sinuses in relationship. I have seen one specimen in which the condition was bilateral. The orbital process of the palate bone articulates anteriorly

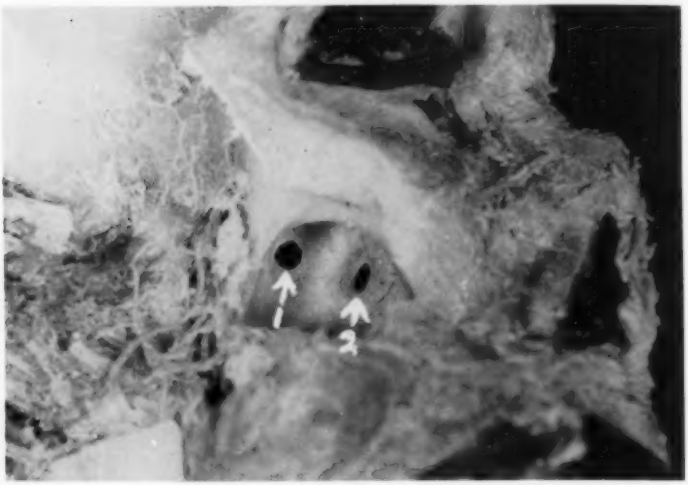

Fig. 5. Usual Area of Contact Between the Palatine Recess of the Right Sphenoid Sinus and the Right Antrum—1; as Related to the Natural Ostium of the Antrum—2.

with the superior maxilla, posteriorly with the ossiculum of Bertini, and medially with the ethmoidal labyrinth. This process may be pneumatized by either the sphenoid or maxillary sinus, in which case the two sinuses are in direct apposition at the antero-lateral-inferior angle of the former and the postero-superior-medial angle of the latter (see Fig. 5). The intervening wall betwen the two sinuses is usually quite thin and may be dehiscent. The lateral boundary between the main sphenoid sinus and the anterior recess appears as a vertical bulge, and is called the sphenopalatine

pillar. This pillar may also be seen when the sphenoid sinus extends into the posterior ethmoid area (see Fig. 3). In some instances the orbital process may be invaded by the posterior ethmoid cell normally related to the lateral half of the anterior sphenoidal wall, and when this situation prevails, the posterior ethmoid cell intervenes between the sphenoid and maxillary sinuses in the nature of a tampon (see Fig. 6). This relationship was first described by Sieur and Jacob.

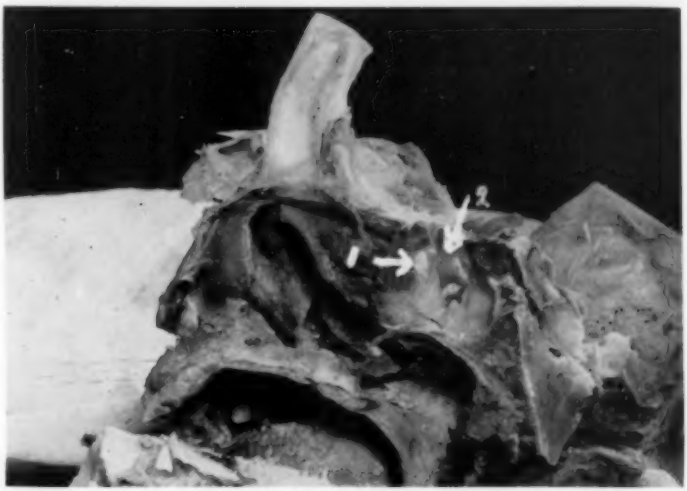

Fig. 6. Right Posterior Ethmoid Cell—1; Between the Right Sphenoid Sinus—2; and the Right Antrum. The Sphenoid-Ethmoid-Antral Relationship was Established at the Tip of the Arrow on No. 2.

Lateral Recess (Great Wing)—When the sphenoid sinus escapes the confines of the body of the sphenoid bone laterally, it may invade the orbital surface of the great wing in the antero-lateral direction and thus bound the orbit postero-laterally (see Fig. 7). This also brings the sinus into relation with the structures passing through the lower part of the superior orbital fissure. Extension of the sinus into the great wing in this area, is usually more extensive than in

other portions of the great wing, because of the resistance to pneumatization offered by the additional fusion planes posteriorly between the alisphenoid, lingula, and basisphenoid.

When the sinus extends more directly laterally, it rarely reaches beyond the great foramina of the middle fossa, but may extend as far as the infra-temporal crest. This brings the sinus into relation with the maxillary division of the Vth

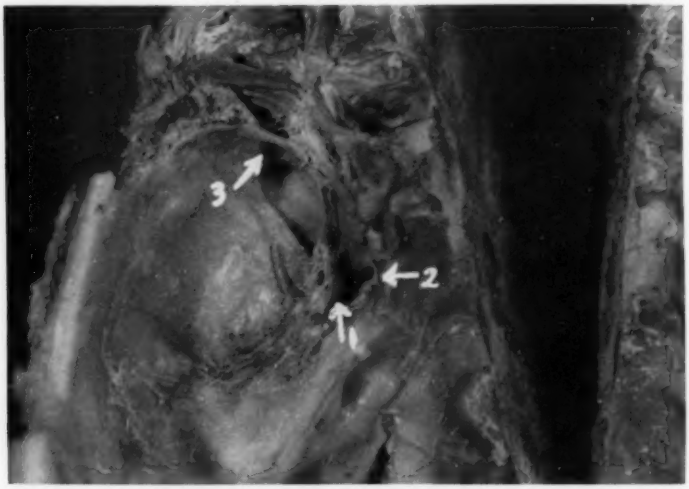

Fig. 7. Large Lateral Recess of the Left Sphenoid Sinus Extending into the Great Wing. Note Relation to the Semilunar Ganglion, Mandibular, and Maxillary Nerves—1; Petrous Apex—2; and Orbit—3.

nerve fairly commonly, and rarely with the temporal lobe of the brain as described by Schaeffer (see Fig. 7).

Pneumatization of the great wing in the postero-lateral plane brings the sinus into relation with the mandibular nerve, Gasserian ganglion, and petrous apex. The relationship, however, is rarely as intimate as that shown in Fig. 7, since invariably there is a wedge of bone intervening between the sinus on the one hand and the above structures on the other.

Fig. 8. Extensive Dehiscence of the Left Bony Vidian Canal—1; the Vidian Nerve is Covered Only by Mucosa Except Below; Accessory Septum—2; Pterygoid Recess—3.

The lateral recess may be bilateral. In one instance I saw the sphenoid sinus of one side form a lateral recess in the great wing of the opposite side. This is uncommon.

Pterygoid (Inferior Lateral) Recess—The pterygoid recess is established by excavation of the base of the pterygoid process (see Fig. 8). It is usually but not always associated with a lateral recess in the great wing of the same side. The condition also is usually unilateral, or at least more pronounced on one side. If the pterygoid plates are invaded the sinus may become related to the antrum in the region of its posterior-inferior-lateral angle; furthermore, if the bone is thin and there is a low origin of the tensor veli palatini muscle on the medial pterygoid plate, the sinus comes into relationship with the Eustachian tube, as shown by Sluder.

Basilar (Posterior) Recess—Projection of the sphenoid sinus posteriorly and inferiorly results in an invasion of the basilar process of the occipital bone, and this may be extensive enough to bring the sinus into close relationship with the foramen magnum. The statement is often seen in the older literature that the more vertical the plane of the basilar process, the more extensive the degree of pneumatization. This has not been verified in my experience. Extensive pneumatization of the basilar process brings the sinus into relationship with the pons, medulla, basilar artery, transverse occipital and inferior petrosal sinuses posteriorly.

Most specimens showing this anomaly also show some degree of absorption of the dorsum sellae. Rarely, when this is complete, the posterior clinoid processes are excavated and the sinus approaches the oculomotor nerve medially and superiorly, and the trochlear nerve laterally at the tip of the process (see Fig. 9). The lateral margin of the dorsum sellae forms the medial margin of Dorello's canal, and is notched for the passage of the abducens nerve. A pneumatized dorsum sellae is related to the abducens nerve in this area.

DEHISCENCES.

Congenital dehiscences may be seen at times on any of the

walls of the sphenoid sinuses, but are perhaps more common on the lateral wall where they may overlay the cavernous sinus or bulge formed by the internal carotid artery, the optic nerve, and more rarely the maxillary nerve (see Fig. 10). They may appear on more than one wall of the same sinus in the same specimen. Dehiscences, in my experience, are

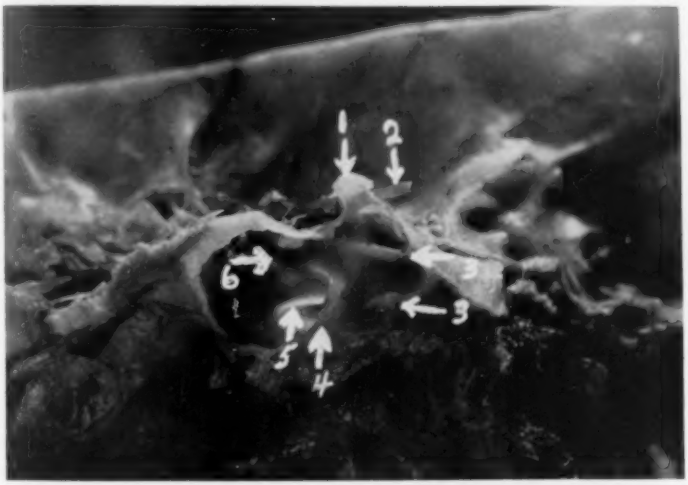

Fig. 9. Pneumatized Dorsum Sellae and Right Posterior Clinoid Process—1; Oculomotor Nerve—2; Horizontal—3; Rare, and Vertical—4; Common, Accessory Septa; Bulge of Maxillary—5; and Ophthalmic—6; Nerves with a Lateral Recess into the Great Wing Below the Maxillary Bulge.

usually quite small and unilateral. Dehiscences overlying the ophthalmic and mandibular divisions of the Vth nerve and the Gasserian ganglion are most uncommon. Schaeffer described three specimens with dehiscences on the lateral wall containing mucosal diverticulae into the cavernous sinus.

Meyer found two specimens each with a similar unique dehiscence in the lateral wall. This anomaly had not been described before, and I do not believe that it has been described since. In both specimens in the lateral wall at the anterior superior angle just beneath the roof was an oval

Fig. 10. Dehiscence in the Lateral Wall of the Right Sphenoid Sinus Overlying the Maxillary Nerve—1; Carotid Artery Dissected—2; Bony Vidian Canal—3.

defect in the horizontal plane. Through this defect the sinal mucosa protruded for a distance forward and upward into a triangular space bounded by the optic nerve antero-medially, the carotid artery postero-medially, and the dura laterally. The sac protruded into the subdural space and could easily be inverted into the sinus. The dura contacted but was not adherent to the margins of the bony defect. In one of the specimens the posterior root of the lesser wing was absent on the side of the defect. The defect described by Meyer corresponds in location to the dehiscence, shown in Fig. 11.

Fig. 11. Congenital Dehiscence in the Lateral Wall of the Left Sphenoid Sinus in the Region of the Infra-Optic Recess. Oculomotor Nerve—2.

On the anterior wall a dehiscence laterally in the wall common to the sphenoid sinus and last posterior ethmoid cell may exist, and if there is a palatine prolongation with a sphenoid sinus-antral relationship, there may be a dehiscence in the wall common to these two sinuses, so that they communicate one with the other.

A dehiscence in the roof of the sinus exposes the pituitary gland if the sinus extends posteriorly to the region of the sella

turcica. Zuckerkandl is reputed to have found a specimen in which the roof of the sinus was completely absent.

A defect in the posterior wall may expose any of the structures lying immediately adjacent. Sieur and Jacob described an interesting specimen in which there were multiple holes in the posterior wall, and extending from this wall forward to the optic groove along the side of the sella turcica was a linear dehiscence containing a large vein.

A bony defect in the floor of the sinus may overlay the Vidian nerve (see Fig. 8).

BULGES INTO THE SINUS WALLS.

Anterior Wall—When a posterior ethmoid cell extends into the sphenoid bone to a slight degree, it causes a bulge on the lateral half of the anterior wall of one or both sphenoid sinuses without appreciable displacement or influence on the development of the sphenoid sinuses. When there is a palatine prolongation extending to the antrum, the anterior wall of the prolongation may appear as a bleb-like bulge into the superior-medial-posterior angle of the antrum.

Lateral Wall—In the region of the antero-superior-lateral angle of the lateral wall absorption of bone by the sphenoid sinus results in the establishment of supra- and infra-optic recesses about the optic canal and prominence of the bulge, formed by the optic nerve either unilaterally or bilaterally. When these recesses are extensive the optic nerve may be virtually completely encircled. The optic bulge may be detected in a posterior ethmoid cell when this cell invades the sphenoid bone. The optic nerves of both sides may bulge into the walls of one sphenoid sinus when there is a marked disparity in size of the two sinuses (see Fig. 2).

The optic bulge is usually seen in conjunction with a bulge formed by the terminal ascending part of the cavernous portion of the carotid artery, and the two are separated by the infra-optic recess. Posteriorly on the lateral wall a bulge is less commonly made by the first ascending part of the cavernous portion of the carotid (see Fig. 12), but this bulge

is characteristically larger than the former one. These bulges are sometimes bilateral. In addition, the carotid artery of one side may form a bulge in the lateral wall of the opposite sphenoid sinus. In one specimen in this series the intersinus septum terminated in the exact center of a large bulge formed by the first ascending part of the cavernous portion of the carotid artery so that it was related to both sinuses. Quite rarely the entire course of the cavernous portion of

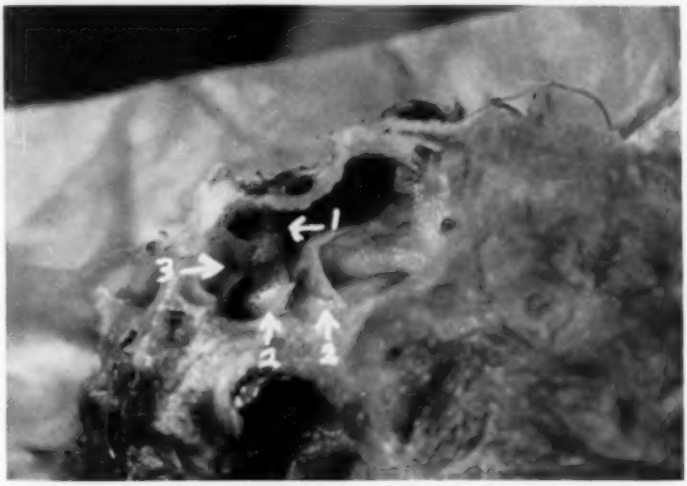

Fig. 12. Bulge of the First Ascending Part of the Cavernous Portion of the Left Carotid Artery—1; Accessory Septa—2, 3.

the carotid artery may be outlined on the lateral wall of the sphenoid sinus (see Fig. 13). This bulge usually exists independent of any lateral recess into the great wing. I have seen three such specimens, and all were unilateral.

Superiorly on the lateral wall one may rarely see a bulge formed by the ophthalmic division of the fifth nerve, and just below this bulge more commonly one may see the prominence created by the maxillary division of the fifth nerve enroute to the foramen rotundum (see Fig. 9). The former is usually unilateral but the latter may be bilateral. The

maxillary prominence is almost invariably associated with a fairly extensive lateral recess into the great wing which passes below and lateral to the nerve (see Figs. 7, 9). Fig. 10 shows a rare exception. In my experience bulges formed by the mandibular nerve and the Gasserian ganglion on the postero-lateral wall are unilateral and extremely rare, since a wedge of bone usually intervenes between these structures and the sinus cavity.

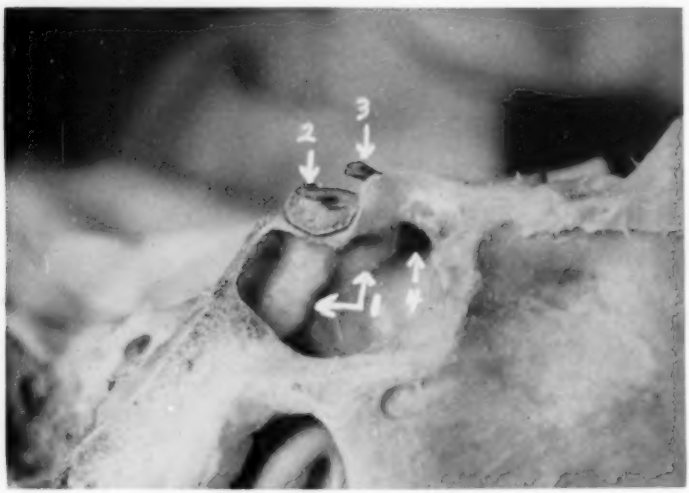

Fig. 13. Cavernous Portion—1; and Cerebral Portion—2—of the Internal Carotid Artery. Optic Nerve—3; Deep Infra-Optic Recess—4. Note the Absence of Other Anomalies of the Lateral Wall.

The nerves of the superior orbital fissure may cause a prominence on the lateral wall anteriorly on either or both sides if the cavernous sinus be short and narrow, as demonstrated by Sluder.

Floor—A projection formed by the sphenopalatine ganglion may be noted in the floor of the sinus anteriorly and somewhat laterally, or in the floor of an encroaching posterior ethmoid cell if one is present in this region. This bulge serves as the roof of the pterygopalatine fossa and is related

to the sphenopalatine ganglion above, and also behind if a pterygoid recess be present. The Vidian nerve projects into the center of the floor of the sinus when the base of the pterygoid process has been excavated by the sphenoid sinus. The condition is usually unilateral or more pronounced on one side (see Fig. 10).

Roof—The pituitary gland encroaches on the roof of the sphenoid sinus to a varying degree when the absorption of the sphenoid bone has advanced posteriorly as far as the pituitary fossa. This projection is most marked when the sinus extends into the dorsum sellae and posterior clinoid processes.

It is my experience, and the experience of others, that the sphenoid sinus is rarely related to the optic chiasm which normally rests on the anterior surface of the pituitary gland several millimeters above and behind the optic groove. The experience of Schaeffer is in contrast to this. In those rare instances when the chiasm does rest in the optic groove, or when it rests low on the anterior surface of the pituitary gland with an associated pneumatization of the tuberculum sellae, the sinus and chiasm may be related.

The structures which throw the walls of the sphenoid sinus into most prominent relief are the cavernous portion of the carotid artery and the optic, maxillary, and Vidian nerves. The abducens nerve does not produce a bulge into the sinus ordinarily; however, it may be intimately related to the sinus as it passes through Dorello's canal, in the superior orbital fissure, and according to Schaeffer, along the lateral wall of the sphenoid sinus when the course of the nerve in the cavernous sinus is occasionally below instead of lateral to the carotid artery.

ACCESSORY SEPTA, RIDGES, CRESTS, AND EXOSTOSES.

Accessory septa are not seen in the rudimentary sinuses which lie anterior to the ossiculum of Bertini-presphenoid plane of synchondrosis; however, a ridge may be present posteriorly representing the anterior end of the fusion line between the body of the sphenoid and lingula. A septum,

ridge, or crest may be seen on the roof, lateral wall, or floor of the sinus at a plane just behind the level of the olivary eminence corresponding to the pre- and postsphenoid fusion line. The most common site for an accessory septum is on the postero-lateral wall, with a small site of origin from the adjacent roof above and floor below (see Figs. 3, 8). This region corresponds to the plane of synchondrosis between the alisphenoid, lingula, and postsphenoid bony nuclei. At times accessory septa, ridges, and crests may appear on any

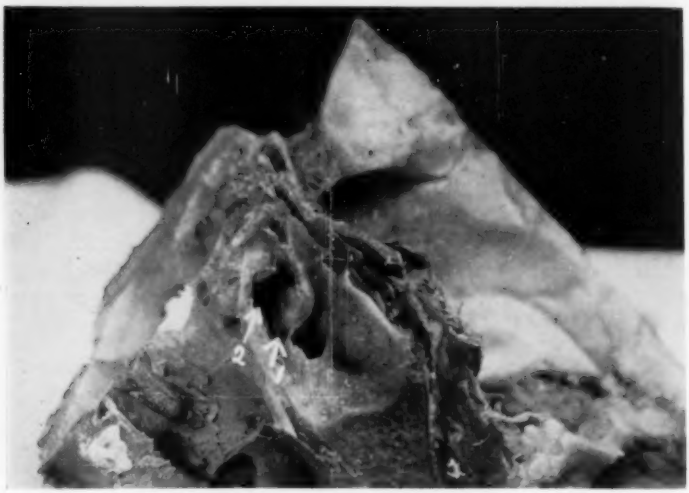

Fig. 14. Accessory Air Cell in Lateral Wall of Right Sphenoid Sinus—1; Palatine Recess—2.

of the walls of the sinus at areas that do not conform to planes of synchondrosis (see Figs. 9, 12). Exostoses are rather infrequent, but in general their presence conforms to fusion lines. The presence of these structures lends irregularity to the contour of the sinus walls and creates extraneous pockets, depressions, and recesses.

ACCESSORY SPHENOID SINUS.

Druss and Grunwald each reported the presence of an accessory sphenoid sinus which existed in the sphenoid bone

without any obvious ostium or connection with the other sphenoid sinuses present. Each author noted his specimen at postmortem examination, and in each instance infection was present. In the case of Druss the accessory sphenoid cavity was posterior to the right sphenoid sinus, filled with pus, and was perforated in the roof. Both of the sphenoid sinuses were inflamed. In Grunwald's case the accessory sinus was filled with pus, but the sphenoid sinuses were normal. The possibility of such an accessory sphenoid

Fig. 15. Pneumatized Lower Root of the Left Lesser Wing—1 Existing as the Only Anomaly of the Left Sphenoid Sinus; Ophthalmic Artery—2; Silhouette of Carotid Artery—3; Semilunar Ganglion and its Branches—4.

sinus in the sphenoid bone is intriguing, since it immediately raises the question of its development and mode of infection if this be present. I have not encountered any other similar reports in the literature. Fig. 14 represents such an accessory air cell encountered in this study. It did not contain the ostium.*†

*This study was made possible through the courtesy of Drs. C. D. Van Cleave and C. H. Hooker, Department of Anatomy, University of North Carolina School of Medicine, Chapel Hill, N. C.

†The photographs were made by Mr. Raymond L. Howard, Durham, N. C.

BIBLIOGRAPHY.

1. Barnhill, J. F.: "The Nose, Throat, and Ear," pp. 104-108, D. Appleton Co., New York and London, 1928.

2. Barnhill, J. F.: "Surgical Anatomy of the Head and Neck," Ed. 2, pp. 236-267, Williams Wilkins Co., 1940.

3. Berger, E.: Variétés de la paroi optico-sphenoïdale. *Bull. et mem. Soc. Franc. Ophthal.*, 12:249-255, 1894.

4. Berger, E., and Tyrmann, J.: Die Krankheiten der Keilbeinhöhle und des Siebbeinlabyrinthes und ihre Beziehungen zu den Erkrankungen des Sehorgans. J. F. Bergmann, Wiesbaden, 1886.

5. Bertemes, G.: "Étude Anatomo-topographique du Sinus Sphénoidal. Applications à la Pathologie des Sinusites Sphénoidales," pp. 1-52, Thèse, de Nancy, 1900.

6. Bonnet, P.: Le Sinus Sphénoidal (étude Topographique). *Rev. Laryngol.*, 53:475-511, April, 1932.

7. Bonnet, P.: Prolongement du Sinus Sphénoidal dans l'Apophyse Clinoide Postérieure, *Bull. Soc. Ophthal., France*, 3:241, March, 1954.

8. Bosworth, F. H.: "A Treatise on Diseases of the Nose and Throat," p. 76, William Wood and Co., New York, 1893.

9. Bruhl, G.: Zur Anatomie der Nebenhöhlen der Nase. *Verhandl. d. Berlin med. Gesellsch*, 31:249-259, 1901; also *Berlin Klin. Wchnschr.*, 37:913-916, Oct. 1, 1900.

10. Budge, E. S.: Anatomical Variations of Accessory Nasal Sinuses. *Calif. and West. Med.*, 33:576-577, Aug., 1930.

11. Caldera: Nouvelles Observations sur Quelques Rapports Anatomiques et Cliniques du Sinus Sphénoidal avec le Sinus Caverneux et les Troncs Nerveux du Moteur Oculaire Commun, du Pathétique, du Trijumeau, du Moteur Oculaire Externe et du Nerf Vidien. *Rev. Hebd. de Laryngol.*, 39:20, Jan. 15, 1918.

12. Caldera: Rapports Anatomiques des Nerfs avec le Sinus Sphénoidal. *Rev. de Moure*, p. 20, 1918.

13. Caliceti, P.: Sulla Esistenza di seni Sfenoidali Multipli e Sulla Loro Importanza Clinica. *Annali di Laringol. Otol., Rinol. e Faring.*, fasc. 3, 1924.

14. Canuyt, G.; Terracol, J., and Leger, V.: Le Canal Optique: Recherches Anatomiques. *Presentation de Pieces, Soc. Anat. de Strasbourg*, Dec. 13, 1923.

15. Canuyt, G.; Terracol, J., and Leger, V.: Études Anatomiques des Sinus Postérieurs. *Presentation de Pieces, Soc. Anat. de Strasbourg*, 1924.

16. Canuyt, G., and Terracol, J.: "Le Sinus Sphénoidal: Anatomie, Exploration, Chirurgie." Masson et Cie, Paris, 1925.

17. Climelli, D.: Contributo alla Morfologia del Seno Sfenoidale. *Oto-Rino-Laring, Ital.*, 9:81-86, Jan., 1939.

18. Congdon, E. D.: The Distribution and Mode of Origin of Septa and Walls of the Sphenoid Sinus. *Anat. Rec.*, 18:97-122, Feb., 1920.

19. Cope, Z. V.: The Internal Structure of the Sphenoid Sinus. *Jour. Anat. and Physiol.*, 51:127-136, 1916-17.

20. Coulouma, P.: Le Sinus Sphénoidal: ses Limites et ses Pro-

longements. *Etude d'Anatomie Medico-Chirurgicale, Echo Med. du Nord.*, 1:1033-1058, July 1, 1934.

21. Coulouma, P.; Bisiaux-Aufort, Mme., and Van Varseveld, F.: Le Prolongement Optico-Alaire du Sinus Sphénoidal. *Echo Med. du Nord.*, 4:732-736, Oct. 27, 1935.

22. Craig, R. H.: Some New Features of the Accessory Cavities of the Nose. *Lancet*, 2:482-483, Aug. 20, 1898.

23. Davis, W. B.: "Development and Anatomy of the Nasal Accessory Sinuses in Man," pp. 160-165. W. B. Saunders Co., Phila., 1914.

24. Davis, W. B.: The Development of the Bones of the Face. *Internat. Jour. Orthodontics*, 3:567-591, Oct., 1917.

25. Davis, W. B.: Anatomy of the Nasal Accessory Sinuses in Infancy and Childhood. *Ann. Otol., Rhinol. and Laryngol.*, 27:940-967, Sept., 1918.

26. Delie: Quelques Démonstrations Anatomiques du Sinus Sphénoide. *Bull. Soc. Belge d'Otol.*, Brux., 6:52-56, 1901.

27. Del Portillo, R., and De Sollano, D.: Senos Esfenoidales. *Pediatria, Espan.*, 19:165-167, June, 1930.

28. Dieulafé, L., and Mouchet, A.: Topographie du Sinus Sphénoidal. *Comp. Rend. de l'Assoc. d'Anat.*, 11:105-113, April, 1909.

29. Dixon, F. W.: A Comparative Study of the Sphenoid Sinus. *Ann. Otol., Rhinol. and Laryngol.*, 46:687-698, Sept., 1937.

30. Dixon, F. W.: Catheterizing the Sphenoid Sinus. *A.M.A. Arch. Otolaryngol.*, 30:994-997, Dec., 1939.

31. Dixon, F. W.: Practical Points in the Treatment of Sphenoiditis. *A.M.A. Arch. Otolaryngol.*, 34:117-120, July, 1941.

32. Douglas, B.: The Pneumatic Sinuses in the Sphenoidal Wings. *Ann. Otol., Rhinol. and Laryngol.*, 10:57-70, Feb., 1901.

33. Douglas, B: "Nasal Sinus Surgery," pp. 153-161. F. A. Davis Co., Phila., 1906.

34. *Druss, J. G.: Intracranial Complications With Disease of the Sphenoid Bone; Report of Two Cases: *1.* Meningitis Secondary to Infection of the Cranio-pharyngeal Canal (?), and *2.* Abscess of the Frontal Lobe. *A.M.A. Arch. Otolaryngol.*, 10:39-48, July, 1929.

35. Durand: Le Sinus Sphénoidal: *Anatomie, Province Med.*, 13:363-366, Aug. 5, 1899.

36. Eadie, C. M.: Posterior Nasal Sinusitis, *Med. Jour. Australia*, 1:487-490, March 12, 1938.

37. *Elfman, L. K.: Dehiscence of Posterior Wall of Sphenoid Sinus; Report of Case. The Laryngoscope, 39:131-133, April, 1929.

38. *Emerson, F. P.: Report of a Fatal Operative Case Showing Developmental Absence of the Outer Sphenoidal Wall and in Its Place a Large Vein Communicating Directly with the Cavernous Sinus. *Trans. Am. Laryngol., Rhinol., and Otol. Soc.*, 14:380-382, 1908.

39. Gaillard, R., et Rollet, J.: Notes sur Quelques Points de l'Anatomie du Sinus Sphénoidal et en Particulier sur sa Vascularisation. *Otol.-Rhinol-Laryngol., Internat.*, 11:569-575, Oct., 1923.

40. Gallemaerts, E.: Nevrite Optique et Sinusite-Sphénoidale. *Ann. Ocul.*, 123:142, Feb., 1900.

41. Gibson, J. A.: The Sphenoidal Sinus; a Study Based on the Examination of 85 Specimens. *J.A.M.A.*, 51:2103-2108, Dec. 19, 1908.

42. Grunwald, L.: "A Treatise on Nasal Suppuration," p. 140, Translated by W. Lamb. Bailliere, Tindall, and Cox, London, 1900.

43. *Grunwald, L.: Intervertebraler Abscess, Ausgehend von einer Eiterung, einer Accessorischen Keilbeinhohle; Zahlreiche Complicationem. *Arch. f. Laryngol. u. Rhinol., Berl.*, 12:454-460, 1901-1902.

44. Haley, J. C.: A Study of the Extent, with Variations, of the Bony Framework of the Paranasal Sinuses. *Med. Rec. and Ann.*, 42:693-696, Dec., 1945.

45. Hansberg: Die Sondirung der Nebenhohlen der Nase. *Monatsschr. f. Ohrenh.*, 24:43-52, 1890.

46. Hanson, W. L.: Embryological Development of the Nasal Accessory Sinuses; Lantern Demonstration. *Illinois Medical Jour.*, 60:386-390, Nov., 1931.

47. Haskin, H.: Presentation of Specimens Showing Probable Etiological Relations of Sphenoidal Sinuses to Some Cases of Tic Douloureaux. The Laryngoscope, 23:710-712, June, 1913.

48. Holmes, C. R.: The Sphenoidal Cavity and Its Relation to the Eye. *Arch. Ophthal.*, 25:460-477, 1896.

49. Houser, K. M.: Anatomic Relation of the Sphenoid Sinus to Dorello's Canal. Abducens Paralysis. *A.M.A. Arch. Otolaryngol.*, 16:488-495, Oct., 1932.

50. Ingersoll, J. M.: A Study of the Development of the Nose and Its Accessory Sinuses. *Trans. Am. Laryngol. Assoc.*, 26:270-291, 1904.

51. Jacob, O.: Prolongement du Sinus Sphénoidal Creusé dans les Grandes Ailes du Sphénoide. *Bull. et Mem. Soc. Anat. de Paris*, 75:403-406, April, 1900.

52. Killian, G.: "The Accessory Sinuses of the Nose and Their Relations to Neighboring Parts," Translated by D. R. Patterson. W. T. Keener Co., Jena and Chicago, 1904.

53. Klepper, J. I.: Anomalous Development of Sphenoid. The Laryngoscope, 37:742-745, Oct., 1927.

54. Laspiur, R. S.: Topografía del Seno Esfenoidal. *Rev. Soc. Med. Assoc., Argent.*, 22:925-950, 1914.

55. Le Double: Sur Quelques Variations des Trous Optiques. *Assoc. de Anat., Nancy*, 4:207-208, 1902.

56. Lee, F. M., and Gibson, J. A.: The Anatomical Relation Between the Sphenoidal Sinus and the Orbit; Observations upon 100 Skulls. *Ophthalmascope*, 9:172-177, March, 1911.

57. Leger, V.: Le Canal Optique et les Sinus Postérieurs. *Thèse de Strasbourg*, 1924.

58. Liu, J. H. and Chang, C. S.: "Paranasal Sinuses in Chinese Infants and Children;" Address Delivered at the Second Congress of the Pan-Pacific Surg. Assoc., Honolulu, Hawaii, Aug., 1936.

59. Loeb, H. W.: A Study of the Anatomic Relations of the Optic Nerve to the Accessory Cavities of the Nose. *Ann. Otol., Rhinol., and Laryngol.*, 18:243-276, June, 1909.

60. Loeb, H. W.: The Cubical Capacity and Superficial Area of the

Sphenoidal Sinus. *Ann. Otol., Rhinol., and Laryngol.*, 21:1-70, March, 1912.

61. Loeb, H. W.: "Operative Surgery of the Nose, Throat, and Ear," Vol. 1, pp. 11-54. C. V. Mosby Co., St. Louis.

62. Maffi, G.: "Seni Sphenoidali: Anatomie e Clinica." G. Bardi, Roma, 1924.

63. Mellinger, W. J.: The Optic Nerves and Their Relations in the Sphenoidal Region. *Trans. Pacific Coast Otol.-Ophthal. Soc.*, 25:85-94, May, 1937.

64. Meyer, A. W.: Perforate Sphenoidal Sinuses With Subdural Diverticula. *Anat. Rec.*, 9:484-490, 1915.

65. Missonici, A. P.: Variations des Rapports Topographiques Entre le Voutre Pharyngée, le Sinus Sphénoidal et la Selle Turcique. *Leur Importance Clinique, Rev. Laryngol.*, 49:331-338, May 31, 1928.

66. Mosher, H. P.: The Anatomy of the Sphenoid Sinus and the Method of Approaching it from the Antrum. The Laryngoscope, 13:177-214, March, 1903.

67. Murphy, J. W.: Head Sections Showing the Relation Existing Between the Nose and Its Accessory Cavities. The Laryngoscope, 11:420-439, Dec., 1901.

68. Mugphy, J. W.: Visual Disturbance Showing a Casual Relation to Disease in the Sphenoid Sinuses with the Report of a Case, Together with Coronal and Sagittal Sections Demonstrating the Relations Existing Between These Cavities. *Trans. Am. Laryngol., Rhinol., and Otol. Soc.*, 13:135-141, 1907.

69. Neivert, H.: Morphologic Variation as a Factor in the Symptomatology of Paranasal Sinus Disease. *A.M.A. Arch Otolaryngol.*, 1:367-383, April, 1925.

70. Nilus, F. and Le Picard, Mme.: Recherches sur le Sinus Sphénoidal et la Selle Turcique chez l'Homme et les Vertebres. *Rev. Franc. d'Endocrinol.*, 11:97-107, April, 1933.

71. Onodi, A.: Die Nasenhöhlen und ihrer Nebenhöhlen, nach Anatomischen Durchschnitten in 12 Talfiein Dargestellt für Aerzte und Studirende. Wien, 1893.

72. Onodi, A.: "The Anatomy of the Nasal Cavity and Its Accessory Sinuses; an Atlas for Students and Practitioners," Translated from Ed. 2, by St. Clair Thompson. H. K. Lewis, London, 1895.

73. Onodi, A.: Das Verhältniss der Kieferhöhle zur Keilbeinhöhle und zu den vorderen Siebbeinezellen. *Arch. f. Laryngol. u. Rhinol., Berl.*, 11:391-395, 1901.

74. Onodi, A.: Des Rapports du Sinus Maxillaire avec le Sinus Sphénoidal et les Cellules Ethmoidales Postérieurs. *Rev. Hebd. de Laryngol.*, 22:177-182, Feb. 16, 1901.

75. Onodi, A.: Des Rapports Entre le Nerf Optique et le Sinus Sphénoidal; la Cellule Ethmoidale Postérieure en Particulier. *Rev. Hebd. de Laryngol.*, 1:721-740, June 20, 1903; also *Arch. f. Laryngol. u. Rhinol.*, 14:360-374, 1903.

76. Onodi, A.: Die Dehiszenzen der Nebenhöhlen der Nase. *Arch. f. Laryngol. u. Rhinol.*, 15:62-71, No. 1, 1903.

77. Onodi, A.: Beiträge zur Kenntnis der Nebenhöhlen der Nase, Pester. *Medicinisch Chirurgische, Presse*, Budapest, 40:558-561, 1904.

78. Onodi, A.: Die Sehstörungen und Erblingdung Nasalen Ursprunges, Bedingt durch Erkrankungen der Hinteren Nebenhöhlen. *Arch. f. Laryngol. u. Rhinol.*, 17:260-274, 1905.

79. Onodi, A.: "Le Nerf Optique et les Cavitiés Accessoires du Nez." A. Holder, Vienna and Leipsig, 1907.

80. Onodi, A.: The Optic Nerve and the Accessory Cavities of the Nose. *Ann. Otol., Rhinol., and Laryngol.*, 17:1-116, March, 1908.

81. Onodi, A.: "Das Gehirn und die Nebenhöhlen der Nase." A. Holder, Wien and Leipsig, 1908.

82. Onodi, A.: Die Eröffnung der Schädenhöhle und Freilegung des Gehirns von den Nebenhöhlen der Nase aus." Curt Kabitzsch, A. Stuber's Verlag, Wurzburg, 1911.

83. Onodi, L.: Ueber die Beziehungen der Keilbeinhöhle zu den Nervenstämmen des Oculomotorius, Trochlearis, Trigeminus und Abducens. *Arch. f. Laryngol. u. Rhinol.*, 26:357-365, July 12, 1912.

84. Onodi, L.: The Rhinogenic and Otogenic Lesions of the Third, Fourth, Fifth and Sixth Cranial Nerves. *Jour. Laryngol. and Otol.*, 29:304-311, June, 1914.

85. Oppikofer, E.: Beiträge zur Normalen und Pathologischen Anatomie der Nase und ihrer Nebenhöhlen. *Arch. f. Laryngol. u. Rhinol.*, 17:28-84, 1905.

86. Parker, C. A.: Accessory Air Cells in the Septum Nasi; an Unusual Development of the Sphenoid Sinuses. *Jour. Laryngol. and Otol.*, 25:562, Oct., 1908.

87. Pelaez Villegas, P. L.: Contribución al Estudio de la Anatomie de las Células Ethmoidales y de la Topografía de los Senos Maxilar, Frontal, y Esfenoidal. *Rev. Ibero-Am. de Cien Med.*, Madrid, 2:308-336, Dec., 1899.

88. *Pickworth, F. A.: The Pathology off the Nasal Sinuses and Its Relation to Mental Disorder. *Jour. Ment. Sc.*, 78:672-673, July, 1932.

89. Prentiss, H. J.: Some Variations in the Sphenoidal Sinus Found in the Anatomical Laboratory of State Univ. Iowa. *Jour. Iowa Med. Soc.*, 5:263-268, July 15, 1915.

90. Radoievitch, S.: Anatomie du Canal Vidien. *Jour. de Med. Bordeaux*, 102:107-110, Feb. 10, 1925.

91. Riezanoff, M. M.: Topography and Surgical Importance of the Sphenoid Sinus. *Khirurgia, Mosk.*, 29:117-141, 1911.

92. Riggle, P. P.: Ethmosphenoidal Cell with Agenesis of the Sphenoidal Sinus. *A.M.A. Arch. Otolaryngol.*, 16:532-537, Oct., 1932.

93. Royet: Presentation de deux os Sphénoides Dont les Dispositions, en ce qui Concerne les Sinus Sphénoideaux, Sont très Notablement Différentes. *Lyon Med.*, 104:55, Jan. 8, 1905.

94. Ruppricht, W.: Über Einige Fälle von Pneumatisation der Kleinen Keilbeinflügel Besonders im Bereich des Processes Clinoid. *Ant., Anat. Anz., Jena*, 58:480-496, Oct., 1924.

95. Schaeffer, J. P.: "The Nose and Olfactory Organ." Pp. 175-202, P. Blakiston and Son Co., Phila., 1920.

96. Schaeffer, J. P.: The Sphenoidal Sinus and the Temporal Lobe. *J.A.M.A.*, 76:1488-1489, May 28, 1921.

97. Schaeffer, J. P.: The Clinical Anatomy and Development of the Paranasal Sinuses. *Penn. Med. Jour.*, 39:395-404, March, 1936.

98. Scheier, M.: Contribution à l'Etude des Anomalies des Cavites Accessoires des Fosses Nasales. *Arch. Internat. d'Otol. et de Rhinol. de Laryngol.*, 14:321-332, Sept.-Oct., 1901.

99. Schlungbaum, A.: Keilbeinhöhlen von Grosser Ausdehnung. *Arch. f. Laryngol. Rhinol.*, 16:514-519, 1904.

100. Sieur, and Jacob: "Recherches Anatomiques Cliniques et Opératoires sur les Fosses Nasales et Leurs Sinus," pp. 282-382. J. Rueff, Paris, 1901.

101. Skillern, R. H.: Accessory Sinuses of the Nose, Ed. 4, pp. 369-378. J. B. Lippencott Co., Phila., 1923.

102. Sluder, G.: The Anatomical and Clinical Relations of the Sphenopalatine (Meckel's) Ganglion to the Nose and its Accessory Sinuses. *Trans. Am. Laryngol. Assoc.*, pp. 329-340, 1909.

103. Sluder, G.: Relations of the Sphenoid Sinus to the Cavernous Sinus. *Trans. Am. Laryngol. Assoc.*, pp. 46-48, 1912.

104. Sluder, G.: Some Anatomic and Clinical Relations of the Sphenoid Sinus to the Cavernous Sinus and the Third, Fourth, Fifth, Sixth and Vidian Nerves. *Ann. Otol., Rhinol., and Laryngol.*, 22:725-768, Sept., 1913.

105. Sluder, G.: Further Observations on Some Anatomical and Clinical Relations of the Sphenoid Sinus to the Cavernous Sinus and the Third, Fourth, Fifth, Sixth and Vidian Nerves. *Ann. Otol., Rhinol., and Laryngol.*, 23:755-758, Dec., 1914.

106. Sluder, G.: The Relations of the Sphenoid Sinus to the Eustachian Tube and Their Possible Clinical Importance. *Trans. Am. Laryngol. Assoc.*, pp. 281-287, 1916.

107. Sluder, G.: The Relations of the Sphenoid Sinus to the Semilunar (Gasserian) Ganglion and Their Possible Clinical Importance. *Ann. Otol., Rhinol. and Laryngol.*, 26:537-538, June, 1917.

108. Sluder, G.: The Anatomy of the Sphenoid Fissure. *Ann. Otol., Rhinol. and Laryngol.*, 32:1108, Dec., 1923.

109. Sluder, G.: Two Factors in Catarrhal Deafness: The Lower Turbinate (Technic) and Sphenoidal Sinus (Anatomy). *J.A.M.A.*, 87:1799, Nov. 27, 1926.

110. Sluder, G.: "Nasal Neurology, Headaches, and Eye Disorders," pp. 139-172. C. V. Mosby Co., St. Louis, 1927.

111. Smith, F.: Management of Chronic Sinus Disease. *A.M.A. Arch. Otolaryngol.*, 19:157-171, Feb., 1934.

112. Stanculeanu, G.: Des Rapports Anatomiques entre les Sinus de la Face et l'Appareil Orbito-Oculaire. *Arch. Ophthal.*, 22:121-132, Feb., 1902.

113. Syme, W. S.: Some Points in the Surgery of the Sphenoidal Sinus. *Trans. Roy. Med.-Chir. Soc., Glasgow*, 8:183-185, 1908-1909.

114. Syme, W. S.: An Examination of 240 Skulls with Reference to the Sphenoidal Sinus and the Spheno-Ethmoidal Cell. *Jour. Laryngol. and Otol.*, 25:78-81, January, 1910.

115. Syme, W. S.: The Sphenoid Sinus in Relation to the Optic Nerve. *Jour. Laryngol. and Otol.*, 39:375-380, July, 1924.

116. Toldt, C.: Osteologische Mittheilungen. *Zeitsch. f. Heilk*, Bd. 4, 1883.

117. Tramontano-Guerritone, G.: Pneumatization of the Small Wing of the Sphenoid Sinus with Abnormal Development of the Fronal Sinus. *Atti Accad. Fisiocrit. Siena*, 1:359-368, 1926.

118. Tunis, J. P.: Sphenoidal Sinusitis in Relation to Optic Neuritis. The Laryngoscope, 22:1157-1164, Oc., 1912.

119. Van Alyea, O. E.: Sphenoid Sinus. *A.M.A. Arch. Otolaryngol.*, 34:225-253, Aug., 1941.

120. Van Alyea, O. E.: "Nasal Sinuses," pp. 155-179. Williams and Wilkins Co., Balt., 1951.

121. Van Gilse, P. H. G.: Bijdrage tot de Kennis van de Vorming van de Wiggebeensholte. *Nederlandschtiijdschrift voor Geneeskunde*, 56:839-841, March 6, 1920; also *Arch. f. Laryngol. u. Rhinol.*, 33:440-446, 1920.

122. Van Gilse, P. H. G.: Sur l'Absence du Sinus Sphénoidal. *Rev. Laryngol.*, 41:529-538, Sept. 30, 1920.

123. Van Gilse, P. H. G.: Zur Pneumatization der Keilbeinhöhle. *Ztschr. Hals-Nasen-u. Ohrenh.*, 3:393-396, 1922.

124. Van Gilse, P. H. G.: Ueber die Bildung des Ostium Sphenoidale Anlässlich eines Praparates mit Scheinbar Fehlender Keilbeinhöhle. *Acta Oto-Laryngol.*, 7:192-198, 1924-1925.

125. Van Gilse, P. H. G.: Investigations on the Development of the Sphenoid Sinus. *Jour. Laryngol. and Otol.*, 41:137-144, March, 1926.

126. Van Gilse, P. H. G.: Ueber die Entwicklung der Keilbeinhöhle des Menchen; Beitrag zur Kenntnis der Pneumatizierung des Schädels von der Nase aus. *Ztschr. Hals-Nassen-u. Ohrenh.*, 16:202-298, 1926.

127. Van Gilse, P. H. G.: The Development of the Sphenoid Sinus in Man and its Homology in Mammals. *Jour. Anat.*, 61:153-166, Jan., 1927.

128. Von Mihalkovics, V.: Bau und Entwickelung der Pneumatischen Gesichtshöhlen. *Verhandl. d. Anat. Gesellsch*, 10:44-63, 1896.

129. von Spee, F. G.: "Bardelen's Handbuch der Anatomie des Menschen," pp. 125-148, Verlag von Gustav Fischer, Jena., 1896.

130. Wallis, G. F. C.: Some Observations Upon the Anatomical Relations of the Optic Nerves and Chiasma to the Sphenoid Bone. *Practitioner*, 98:41-52, Jan., 1917.

131. Westlake, S. B.: A Note on the Relation of the Optic Nerve to the Last Posterior Ethmoid Cell. *Ann. Otol., Rhinol, and Laryngol.*, 32:730-748, Sept., 1923.

132. White, L. E.: A Study of the Optic Canal. *Ann. Otol., Rhinol. and Laryngol.*, 33:121-151, March, 1924.

133. Zuckerkandl, E.: "Normale and Pathologische Anatomie der Nasenhöhle und ihrer Pneumatischen Anhänge, pp. 169-173, W. Braumüller, Wien, 1882.

134. Zuckerkandl, E.: "Normale und Pathologische Anatomie der Nasenhöhle und ihrer Pneumatischen Anhänge," Vol. 1, Second Re-edited Edition, W. Braumüller, pp. 335-340, Wien and Leipsig, 1893.

135. Young, G.: The Relation of the Optic Nerve to the Sphenoidal and Posterior Ethmoidal Sinus. *Brit. Med. Jour.*, 2:1258-1259, Dec. 30, 1922.

136. Young, G.: Relations of the Optic and Vidian Nerves to the Sphenoid Sinus. *Jour. Laryngol. and Otol.*, 37:613-617, Dec., 1922.

137. Young, G.: Retrobulbar Neuritis of Sphenoidal Sinus Origin. *Jour. Laryngol. and Otol.*, 39:381-383, July, 1924.

138. Onodi, A.: "Die Nebenhohlen der Nase. Nach Photographischen Aufnahmen 124 Praparte in Naturlicher Grosze Dargestellt." A. Holder, Wien, 1905.

139. Onodi, A., and Rosenberg, A.: "Die Behandlung der Krankheiten der Nase und des Nasenrachens." Verlag von Oscar Coblentz, Berlin, 1906.

140. Onodi, A.: "The Optic Nerve and the Accessory Cavities of the Nose; a Contribution to the Study of Canalicular Neuritis and Atrophy of the Optic Nerve of Nasal Origin," pp. 1-101, Translated by J. Luchkoff. William Wood & Co., New York, 1910.

141. Onodi, A.: "Die Beziehungen der Tranenorgane zur Nasenhohle und zu ihrer Nebenhohlen." Urban & Schwarzenberg, Berlin and Wien, 1913.

142. Catalog of the Onodi Collection, pp. 115-127 (Museum of The Royal College of Surgeons). Headley Brothers, London, 1934.

*Reported as anatomical variations in clinical or postmortem cases associated with infection.

UNIVERSITY OF ILLINOIS.

The next course in Laryngology and Bronchoesophagology to be given by the University of Illinois College of Medicine is scheduled for November 4-16, 1957, under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

ACOUSTIC ACCIDENT*

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By the term acoustic accident, I have in mind the sudden loss of serviceable hearing, typically in one ear, from hearing that was supposedly usable. Although most patients do not have previous records of their hearing, there is always some reason for them to know that they were not deaf in either ear. Repeated ebbs and flows of hearing following acoustic accident are not the rule. The hearing sinks in waves of dizziness and sea shell tinnitus; occasionally all may be calm. There is neither previous warning nor subsequent explanation.

One or more additional inner ear symptoms of tinnitus, nausea, vomiting and vertigo are present in more than half of the cases. It may be true that the greater the loss of hearing, the more severe the vestibular symptoms.

"Meniere-like syndrome", "pseudo Meniere's disease", and "Meniere symptom complex", all possibly incorporating acoustic accident, are terms which act as coveralls for a multiplicity of undiagnosed and self-styled vertiginous complaints, real or considered, in a most indefinite sort of way. They will not be discussed in this writing.

In order to avoid confusion, these cases under the title of Acoustic Accident, must fulfill the following criteria: *1.* the patient is reasonably and intelligently certain that his hearing was conversational in the now affected ear; *2.* the attack under consideration is the first of its kind that the patient has experienced; *3.* with the attack, the hearing in the affected ear is rendered so low that no longer is it fit for normal or amplified conversation although a few tones, manifest in the highest decibel range, may be present.

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DIAGNOSIS.

In the differential diagnosis, two other conditions are introduced: labyrinthitis and Meniere's disease. In labyrinthitis, one thinks of an obvious reason, a veritable cause, an infection-type of illness. Although an infection can (and has happened) be concealed in the internal ear, this is not often the case. It is most likely to follow middle ear or mastoid disease, which is suggestive.

Meniere's disease is more definite, if one adheres to the typical triad of impaired hearing, tinnitus and vertigo. It is a disease with repeated episodes; a disease in which the hearing is decidedly lowered during the attack, but without complete loss of some or all tones in the affected ear; a disease in which the hearing returns to near normal between attacks. The vestibular reactions follow those of the cochlea; that is, they are hypoactive in the early part of the attack and normally active between attacks. This is not an unequivocal rule, for the actively diseased labyrinth may be irritable, causing a hyperactive response; but the differential point is the return of cochlear function.

The cause of acoustic accident is not known, unless one wishes to include that occasional case which follows mumps. Evidence of infection, or a possible source of infection, is lacking. One attack eradicates practical hearing. In other words, acoustic accident strikes; but, having struck, it remains.

It is evident that some reliance is placed on the caloric test in spite of the general feeling that the caloric test provides only two bits of fruitless information; that the labyrinth does react, or does not; however, if a caloric test should be carried out with relatively warm water, for example, 80°F., for one minute, there should be a good chance of picking up lesser labyrinthine reaction differences.

As an example of acoustic accident may be cited:

Mrs. S. P., a 66-year-old motel owner, stated that she had never had any physical or mental disability more than delivering four children, two presently alive. She was certain that she could hear conversation in each ear until one night a few weeks before her examination on April 2, 1956. She had just finished the evening meal duties when

she became violently dizzy. Fifteen minutes later, she began to vomit. During the five days that she spent in the hospital, she could not walk without help. From the beginning of the attack, she noted that the left ear was "dead." In her case, there was no tinnitus, and dizziness was general throughout her head. When she was examined in my office, the audiogram disclosed a few valueless tones in the left ear and only fair hearing in the right ear. The caloric tests were somewhat comparable to the audiometric tests; that is, the left inner ear responded poorly to the caloric irrigation while the right inner ear responded normally.

The diagnosis was made on the basis that in acoustic accident, there is loss of practical hearing in the affected ear following one attack, and a decrease in the labyrinthine response to caloric stimulation which generally is not so profound as that of the hearing, although there are cases in which labyrinthine response cannot be elicited by irrigating with ice water for two minutes. Acoustic accident does not have the distinctive *Meniere's rebound* of cochlear and labyrinthine functions.

STATISTICS.

Thirty-four patients were seen from January, 1949, to August, 1956. Ten were male, 24 female. The right ear was affected 22 times, the left ear 12. There were two patients in the 10-20 year age group; four in the 20-30; five in the 30-40; ten in the 40-50 (often termed by the patients the "falling-to-pieces" decade); four each in the sixth, seventh, and eighth decades, and one in the ninth. The youngest was a 12-year-old female, who had total loss of demonstrable hearing in the right ear. She stated that there was no question about having had hearing in that ear, for she had always used it on the telephone. After a brief illness during which she had nausea, vomiting and vertigo, she thought that the telephone was out of order until she changed to the other ear. The oldest was an 85-year-old man who awoke one morning thinking that he had a "July fly" in his right ear. He interpreted the slight vertigo as biliousness; but, he was unable to explain why he did not hear through his noisy ear.

In 30 of the 34 cases, the deafness was realized suddenly; in the other four, it came on within a period of two weeks.

In addition to the unilateral deafness, 23 had nausea, vomiting and vertigo in varying amounts, and 18 were annoyed with tinnitus; only 11 had deafness alone.

Pain appeared twice, once as an earache, the other time as pain in the head, back, arms and legs, for which no cause could be found. There were no relevant positive findings, except the otologic, in any patient.

Sixteen were tested for labyrinthine function by the caloric method. Each had normal response in the unaffected ear. Twelve displayed reactions varying from minimal to no response in the affected ears. The labyrinths of each of four patients reacted equally. In no case did the deafened ear exhibit a hyperactive labyrinthine reaction. It should be remembered that these tests were not run at the same post-attack time. Indeed the amount of time from the original attack to examination in my office varied from 15 hours to 12 years. Of those not tested, a frequent reason was the patient's decision.

Eleven patients had temporal bone X-ray studies. There was no evidence of infection, tumor, or anomaly.

TREATMENT.

Not all patients were treated. Where the deafness had been present for a long time, even years before examination, the initial findings were regarded as final. Those who did receive treatment were usually persons whose attacks were recent. The hearing of two patients improved, one after two weeks of treatment, the other in approximately one month but neither patient regained conversational hearing. Medication consisted of Vastran, saturated solution of Potassium Chloride, and Cortril in the first case, and Vastran alone in the second. This information afforded a bit of optimism until further search revealed that eight patients were treated without help. Seven of these received the above mentioned drugs, plus Parenzyme, Nicotinic Acid, Aureomycin, Elixir Pyribenzamine, Protamide, Viprotinal and Dramamine. The eighth patient presented himself 15 hours following the onset of his unilateral total deafness. He was hospitalized

and treated for five days with vasodilating medication and anticoagulants. Following hospitalization, he was continued on Vastran for six weeks. He remained totally deaf in that ear when dismissed after 12 weeks of observation. The two patients who gained hearing were a 26-year-old male and a 36-year-old female: the male did not have any cochlear or vestibular symptoms except deafness, and his hearing improved in two weeks; the female had deafness, nausea, vomiting and tinnitus, but no vertigo, and her hearing was better in one month. In each, the labyrinthine responses were equal and considered normal for both ears on first examination.

Two patients, not numbered among these 34 because of difficulty of classification, showed hearing gain in one month, only to revert to the poor hearing after another month, and being under treatment all the while.

THEORY.

There was no evident cause for the attack, unless it was in the three patients who had mumps: in one, the attack occurred at the onset of mumps; in the second, as the patient was recovering; in the third, nine days after the patient was considered well. Aside from these three, the most consistent clue to a cause, and this infrequent, was the occasional patient's statement that although the ear had been usable, it had not been regarded as being as good as the other ear for a long time. This usually meant several years. Could it be possible that a defective VIII nerve end organ would be more unstable?

Acoustic accident is not considered to be a recurring condition, as regards the hearing, probably because the hearing does not improve enough to fail again. Occasionally, however, a patient will have recurring vertiginous attacks, even though the cochlear and labyrinthine functions remain depressed. For that, labyrinthotomy is well advised. Only one person in this group required operation. The soft tissue which was removed from the vestibule was prepared for

microscopic examination. This failed to show evidence of hemorrhage, and the few cells that were seen were thought to be inflammatory.

Since establishing a cross file for acoustic accident in 1948, I have found that these cases become more common as one becomes more interested. In fact, if one but knew how to classify this condition, he might find that there are varying degrees of severity. For example, consider the person who suddenly has a feeling of one ear's being partially stopped up, possibly accompanied by some ear noise and even slight vertigo. Gross examination discloses a normal appearing ear. The audiogram may show a decrease of hearing, especially in the high tones, but there is retention of amplified conversational hearing. From the few such cases that I have followed, it would be my impression that these stand a better chance for recovery, although the hearing may not become completely normal.

LITERATURE.

From the literature of 1950, come articles by Fowler[1] and Lindsay.[2] Fowler found that emotional stress occurred often in his cases; also, he considered the possibility of blood sludge as a causative factor. Of his 26 patients, 75 per cent were male. He quoted from Rasmussen's[3] 18 cases, of which 83 per cent were female. Lindsay observed that the labyrinthine responses in the two ears with the "cold caloric" test were normal and equal in half of his 16 cases. He suggested vascular disturbance for etiology. These men referred to the condition as "sudden deafness" and "inner ear deafness of sudden onset." Carco's[4] article on "Cochlear Block" referred to Citelli's[5] article of 1926, in which he described sudden deafness without vestibular symptoms as *always* following exposure to cold. Carco hedged a bit, stating that a *majority* were caused by exposure to cold, and that not all were free from vestibular disturbances. One of the most impressive literature findings was that Fowler had seen cases of bilateral "total deafness" from this or a related condition.

ADDENDUM.

In almost every instance, the patient had some explanation for his plight. None could be substantiated scientifically. There was no uniformity as to time, place or patient's action regarding the onset. One patient had attended a party the night before. Having dieted on crabmeat, beer and cocktails, he was not surprised to awaken the next morning with nausea and vomiting, but he was at loss to decide which ingredient caused him to be deaf in one ear. Another patient stated that he had had indigestion as long as he could remember, but none since his ear "went out." A third patient stated that when she suddenly went deaf in one ear, she thought that someone had struck her on the head. In each of three cases, the patient was deaf immediately after returning from a trip to the mountains. One patient was watching the T-V news of California floods. Her interest was running high because of having a son in the area. At the peak of tenseness, "something went zoom," and the left ear was deaf. She interpreted this as a break in the tenseness. To say the least, it broke her line of thought.

SUMMARY.

1. Acoustic accident is a condition in which one ear suddenly, permanently, and without explanation becomes deaf, with or without vestibular symptoms. There is regularly a depression of vestibular response to mild caloric stimulation.

2. It is differentiated from Meniere's disease mainly by its lack of the typical "Meniere's rebound."

3. The cause is unknown. In the one case where the contents of the vestibule were microscopically examined, there was no evidence of hemorrhage.

4. Occasionally, the patient mentions that although the hearing previously was serviceable in the now impaired ear, it was not thought to be as good as that in the other ear.

5. The beneficial results of treatment are doubtful. Labyrinthotomy is necessary for repeated attacks of labyrinthine vertigo.

6. Bilateral acoustic accident has not been seen by the author in these eight years.

REFERENCES.

1. Fowler, E. P.: Sudden Deafness. *Ann. Otol., Rhinol. and Laryngol.*, 59:980-987, March, 1950.

2. Lindsay, J. R.: Inner Ear Deafness of Sudden Onset. *Trans. Fifty-fourth Ann. Meet. Amer. Laryngol., Rhinol. and Otol. Soc., Inc.*, 94-123, 1950.

3. Rasmussen, H.: Sudden Deafness. *Acta Oto-Laryngologica*, 37:65-70, Feb., 1949.

4. Carco, P.: Sull'Interpretazione Patogenetica del Cosidetto Blocco Cocleare. *L'Oto-Rino-Laringol., Ital.* (Bologna) Vol. XX, 520-529, 1952.

5. Citelli, S.: *L'Oto-Rhinol.-Laryngol. Internat.*, 1926.

490 Peachtree Street, N. E.

NATIONAL HEARING WEEK, MAY 5-11, 1957.

Because it is not a "visible" handicap, the average American is not aware that nearly one in ten of his fellow citizens suffers from some degree of hearing loss. That of these estimated 15 million hard of hearing persons some three million are young children is often not apparent even to parents and families who think their youngster is just "slow to talk" or "not paying attention."

The American Hearing Society, during its Twenty-ninth Annual National Hearing Week hopes to alert the public to the problems of hearing loss and the importance of efforts to prevent deafness, conserve hearing and, failing those, then rehabilitation.

To help those facing a lifetime of silence in our sound-filled world, send your gift to:

The American Hearing Society,
1800 H Street, N. W.,
Washington 6, D. C.

NONCHROMAFFIN PARAGANGLIOMA OF THE NASAL SINUSES.*†

WILLIAM BLAKE HARKINS, M.D.,

St. Louis, Mo.

There are numerous small structures widely distributed throughout the body, consisting of multiple vascular channels surrounded by epithelioid cells which lie in nests, cords and sheets between the vessels; they are well-defined, encapsulated bodies, separated into lobules by fibrous septa. These structures include the carotid body, the aortic body, the ciliary paraganglion in the orbit, the glomus jugulare and the glomera of the skin of the extremities and of deeper structures of the body.[2,22] Although they have been spoken of as a part of the chromaffin system, it is now believed that they are not glands of internal secretion such as the adrenal medulla.[14,22] These bodies, or glomera, have these characteristics in common: close association with ganglia of sympathetic nervous system and intimate association with blood vessels. They are believed to be special chemo-receptor bodies, concerned with the reaction of the blood.[5,11] Tumors arising from these bodies, or glomera, have come to be known as non-chromaffin paraganglioma, a term first proposed by Lattes in 1950.[13]

The case of a 59-year-old woman is reported, in which there occurred a rather large tumor involving the nasal sinuses and nasal fossa. This tumor was observed and studied, intermittently, over a period of four years. The patient gave a history of nasal obstruction and rhinorrhea of at least 30 years' duration, which she had been told were due to nasal allergy. In 1927 she had had nasal polyps removed. Polyps were again removed from the nose in 1944, 1948, and 1952. At the time of the last polypectomy (1952), which

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was done in Lincoln, Neb., the operator noted an unusual amount of bleeding from the left nasal passageway. Tissue removed from the left side of the nose, at that time, was reported by the local pathologist to be a malignant tumor, a carcinoma of mucous gland epithelium; however, the pathologist expressed uncertainty as to his own diagnosis.

Soon afterward the patient went to the Mayo Clinic and took sections of this tissue with her. The pathologists at the Mayo Clinic examined the sections and made a diagnosis of "chemodectoma", which they stated meant a neoplasm similar to a carotid-body tumor. They recommended only excision of the tumor locally, within the nose whenever it recurred, advising against the use of radiation. They stated that these tumors do not respond well to irradiation, and they did not believe any radical surgical excision was indicated, as there would be no assurance that the lesion could be entirely eradicated.

This patient first came under my care in the autumn of 1954, at which time the nasal cavities presented only the classical picture of allergic rhinitis: pale, edematous mucosa, multiple, pale gelatinous polyps; and copious clear mucus which showed many eosinophiles. X-rays of the nasal sinuses at this time showed only a mild, generalized haziness of the left maxillary sinus and left ethmoid cells; there was no bone destruction (see Fig. 1).

On Nov. 4, 1954, at DePaul Hospital in St. Louis, I removed polyps from both nasal fossae, by means of snare, and discovered the tumor. I had an extremely limited view of it; the lower edge of a rounded moveable mass, in the left choana, looking like a reddish-purple polyp of firm consistency. It was possible to remove only a piece of this tissue; there was free bleeding, which was self-limited.

The tumor tissue was examined by the pathologist at DePaul Hospital who stated that it was either an anaplastic carcinoma or a hemangio-endothelioma. These sections were submitted to the pathology department of St. Louis University School of Medicine, and we obtained a diagnosis of glom-angioma.

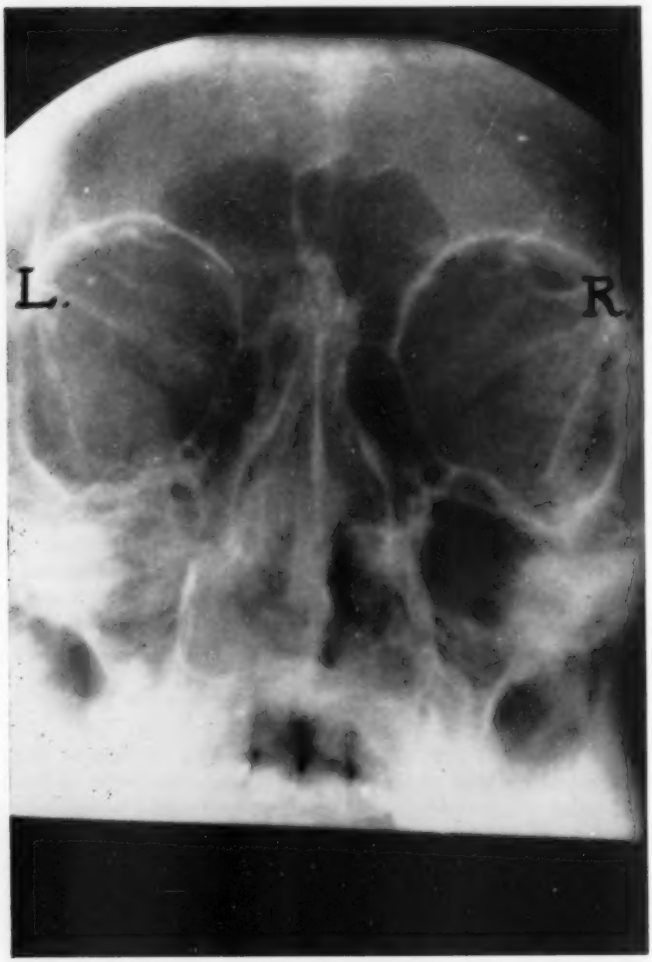

Fig. 1. Roentgenogram of nasal sinuses in Nov., 1954, showing only a mild degree of clouding of left maxillary sinus and left ethmoid cells; no bone destruction.

The patient was fairly comfortable for the next 14 months, with only occasional nasal obstruction and a mild rhinorrhea; but by mid-December, 1955, nasal obstruction had become constant, and she began to bleed from the left nasal fossa. Thereafter the bleeding became more frequent and profuse. Posterior and anterior nasal packing finally controlled the epistaxis, and on Jan. 14, 1956, she entered St. Mary's Hospital.

The physical examination on admission to the hospital was not remarkable, except for the nose and throat. The nasal mucosa was pale and edematous, and it was possible to obtain just a glimpse of a deep purple mass in the left choana. X-rays of the nasal sinuses showed a homogenous area of increased density over the left ethmoid and antrum, with absorption of the bony architecture of the left nasoantral wall. The remaining sinuses were clear (see Fig. 2).

Dr. James B. Costen saw this patient in consultation. His clinical impression was "hemangio-endothelioma", and he advised radical surgical excision of the tumor after ligation of the left external carotid artery, and suggested a course of deep X-ray therapy, both before and after surgery.

On Jan. 21, 1956, a preliminary ligation of the left external carotid artery was carried out. On Jan. 24, 1956, the tumor was removed through a lateral rhinotomy on the left side. We encountered a lobulated tumor mass, estimated at approximately 7x7x5 cm., having the general contour of a fat mushroom. There was a very short pedicle or stalk, about 1 cm. in diameter, arising in what apparently had been a posterior ethmoid cell on the left side. The tumor mass protruded from the left ethmoid region and completely filled the entire left nasal cavity; it obliterated and filled all of the ethmoid cells; had eroded the anterior wall of the sphenoid sinus and filled this cavity; had eroded the lamina papyracea; the periorbita was exposed but not invaded. The nasoantral wall was completely destroyed by the tumor, which had extended into and filled the left maxillary sinus. The floor of the orbit was intact; the anterior wall of the antrum was very thin from the pressure of the tumor, but was intact.

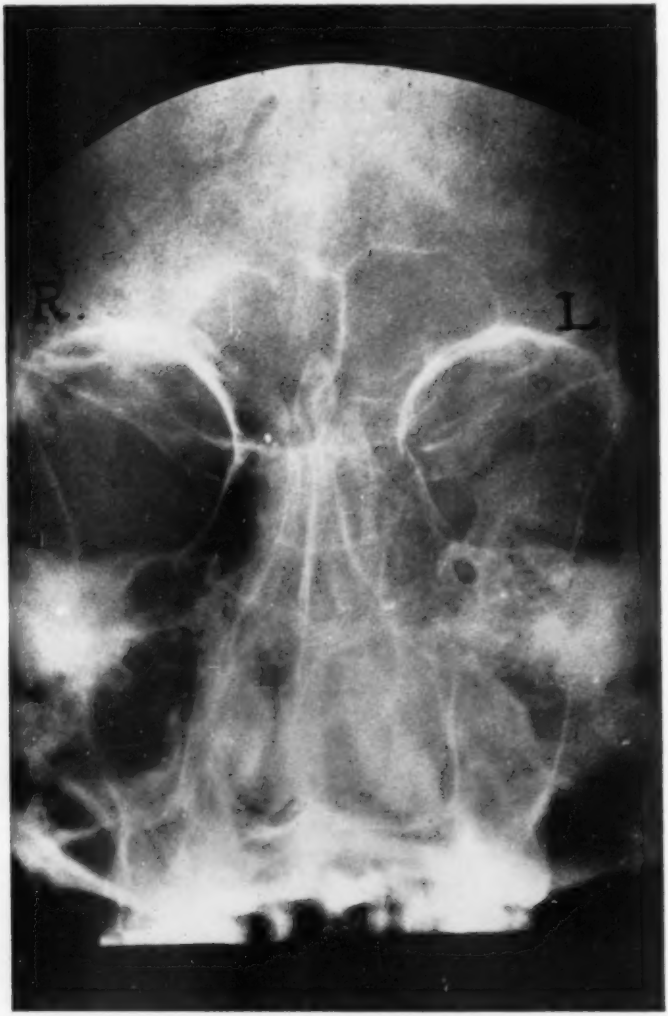

Fig. 2. Roentgenogram of nasal sinuses in Jan., 1956, showing increased density of left ethmoid cells and left maxillary sinus, with absorption of left nasoantral wall.

There were no turbinates remaining in the left nasal cavity, except a small stump of the anterior part of the inferior turbinate. The tumor had pushed its way through the upper posterior portion of the septum and presented a small lobule of tumor tissue in the right side of the nose. The tumor had a deep cyanotic purple-red color. It showed a smooth, slick surface. I emphasize the fact that the tumor had a definite capsule. It was lobulated and was not attached to the structures with which it came in contact; showed no invasion, but apparently forced its way through all adjacent structures by pressure. Bleeding was quite profuse wherever the neoplasm was touched. The tissue had a spongy, friable consistency, and reminded us of nothing so much as placenta.

It was an easy matter to deliver this tumor, as it had no attachment except the stalk in the posterior ethmoid area; however, it was so friable that it broke into fragments as removed. After the tumor had been removed the entire cavity was packed firmly with gauze, brought out through the left nostril. The incision was closed, and a firm pressure dressing was applied to the left side of the face.

We were fortunate enough to obtain primary union of the soft tissues of the face. There were no disturbances in the motility of the left eye-ball. All nasal packing was removed on the sixth post-operative day.

After healing we started radiation therapy, under the guidance of Dr. L. R. Sante, directed to the left posterior ethmoid area, where we felt that there might possibly be tumor tissue at the attachment of the stalk. The patient received 200r. per treatment, given daily, through two parts (an anterior and a lateral) for 12 days, for a total of 2400r. Mild hyperemia and edema of the left upper and lower eyelids appeared but subsided promptly after completion of the X-ray therapy. Subsequently there has been a mild degree of atrophy of nasal mucosa, with a tendency to the formation of crusts. The patient irrigates this cavity once or twice a week. We are able to visualize this entire cavity easily through the left nostril. The site of the attachment at first had a suspicious purple color, but has gradually faded out to the usual pink color of nasal mucosa.

The pathologists at St. Louis University (of which St. Mary's Hospital is a part) studied the sections of the tumor and also conferred with Dr. L. V. Ackerman of the Department of Pathology of Washington University School of Medicine, and made a diagnosis of olfactory esthesic-neuroepithelioma. In their report they made reference to a similar case at Barnes Hospital, St. Louis, reported by Seaman in 1951.[19]

The sections (see Figs. 3 and 4) showed collections and nests of cells, separated by fibrous septae. The arrangement of the cells did not show any pattern; no rosettes nor acini. Most of the cells were rather small, oval, with a faintly eosinophilic cytoplasm and small dense nuclei. There were occasional areas of larger oval cells, with eosinophilic cytoplasm, larger paler nuclei and prominent nucleoli. Mitotic figures were rare. The fibrous septae were variable for the most part, thin and irregular; in some areas, quite dense and showed hyalinization. There were many blood vessels, many of them large, irregular endothelial-lined spaces.

Ackerman and del Regato[1] stated that tumors similar to carotid body tumors arise from the aortic body, from the glomus jugularis and from cells associated with the ganglion nodosum of the vagus. These organs consist of nonchromaffin, non-epinephrine producing bodies with sensory innervation. They are to be distinguished from the adrenal medulla. They probably represent chemoreceptors. Of this group, the carotid body tumors are the commonest. They are slow-growing and have a well-defined capsule. Microscopically the tumor is made up of cell-nests with uniform cells, without mitotic activity. These cell nests are surrounded by a vascular stroma. They are practically never malignant; the chances of metastases are remote. Excision is said to be the treatment of choice, but a few instances of successful radiotherapy have been recorded. Ewing[7] places carotid body tumors under the class of endotheliomas, and we cannot be certain that our case is not an endothelioma.

Data on carotid body tumors given by Ward and Hendrick[20] is essentially similar. Reports of response to irradiation vary, although most reported trials with irradiation

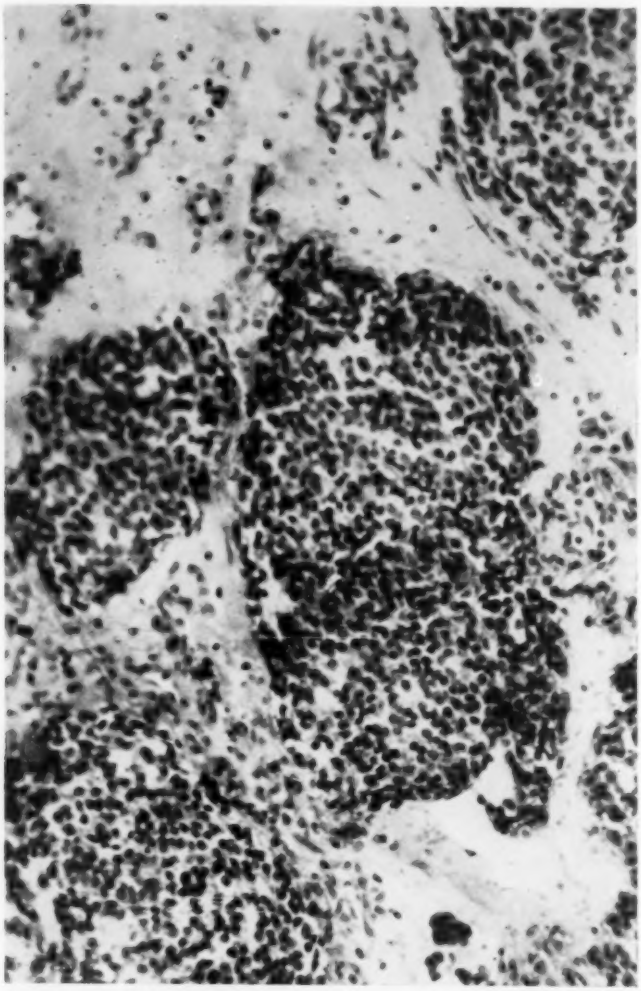

Fig. 3. Microscopic section of tumor, showing nests of cells, separated by fibrous septae. Most of the cells are small, oval, and show small, dense nuclei. There are numerous blood vessels, many of them large, irregular, endothelial-lined spaces. (Low power).

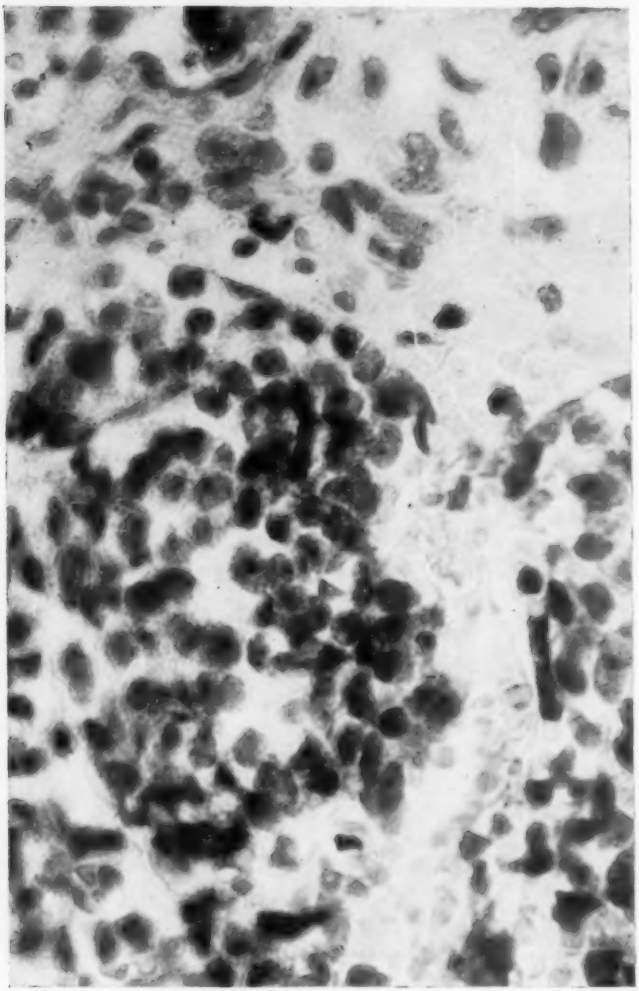

Fig. 4. Same as Fig. 3, under high power.

have been unfavorable. Although usually benign, instances of metastasis have occurred. Romanski[16] had one case of his own and reported 15 other cases from the literature, with metastasis from carotid body tumors.

In 1941 Guild[9,10] described a hitherto unrecognized structure in the temporal bone and called it glomus jugularis. The first tumor of the middle ear arising from the glomus jugularis was recognized by Rosenwasser[17] in 1945. Since then, numerous cases have been reported.[23] The tumor is described as a dark-red or purple, soft polypoid mass, with a tendency to destruction of adjacent structures, either by pressure or by invasion. The tumors are vascular and bleed profusely when incised. Structurally, these tumors are similar to carotid body tumors. There are round or polygonal cells, showing abundant, slightly granular, eosinophilic cytoplasm, and round or oval nuclei with punctate granules of chromatin and small, sometimes multiple, nucleoli. The tumor is highly vascular, and the walls of most of the blood vessels consist of little more than a single layer of endothelial cells. The tumor cells lie in ribbons or nests, in intimate association with the blood vessels. Trabeculae of connective tissue traverse the tumor, producing a lobulated or alveolar pattern.

Williams and his associates[21] presented 13 cases of glomus jugulare tumors; they adopted the name "chemo-dectoma", as first proposed by Mulligan,[15] since that author believed them to consist of chemo-receptor cells which are associated with para-sympathetic nerves and which originate in the adventitia of blood vessels. Williams and his colleagues concluded that surgery alone was not able to prevent the advancement of the lesion. They found a fairly good response to X-ray. Lattes and Waltner[12] called these tumors nonchromaffin paraganglioma.

Recently, Andrews[3] reported a neoplasm having this same histological picture, occurring in the larynx. He called attention to the fact that glomus tumors and glomus bodies are found in many parts of the body: the carotid body, skin of extremities, middle ear, aortic-pulmonary area, ganglion modosum, region of the ciliary ganglion, corpus cavernosum,

glomus coccygeum, intestinal villae, wall of the stomach, knee joint.

It is the opinion of the author that the case herein reported is compatible with the diagnosis of nonchromaffin paraganglioma. It presents the character of a vascular, encapsulated tumor, growing slowly, expanding and pushing its way through contiguous structures, but not invading. Histologically it presents the picture of a glomus jugulare or carotid body tumor, and was so recognized by the pathologists at the Mayo clinic, by the pathologists at St. Louis University School of Medicine on the first examination (in 1954) and by an independent pathologist, Dr. W. J. Siebert of the Lutheran Hospital in St. Louis (verbal report).

The diagnosis we received from the pathologists of St. Louis University School of Medicine when the entire tumor was submitted, in 1956, namely "estheric-neuro-epithelioma" was confusing. They referred to the case of Seaman,[19] which had a clinical history and histological picture similar to the case presented here. Seaman's case was malignant, and metastasized to the cervical nodes and invaded adjacent muscle. Berger, Luc and Richard[4] first described such a tumor in 1924. It is said to have arisen from the sensory cells of olfactory epithelium. In some of their cases rosettes and neurofibrils were present, and the neoplasms apparently were a type of neuroblastoma, occurring in the nasal fossae. They were extremely radiosensitive, but recurred.

Schall and Lineback[18] reported three cases of primary intranasal neuroblastoma and reviewed the literature on this kind of tumor. Their cases were benign, the neoplasms having the apparance of very vascular, bleeding polypi, originating in the ethmoid cells. There were masses and cords of cells, having indistinct cell-boundaries, separated by capillaries and larger vessels. The tumor cells showed prominent round or oval nuclei, with finely granular chromatin. In one of these cases, there was a pseudo-rosette formation. These authors felt that the tumors in their cases originated from olfactory neuroepithelium and were of the same type as described by Berger, Luc and Richard.[4]

Fisher[8] says that there are neoplasms of the nasal fossae characterized by an undifferentiated neuroectodermal structure identical with that observed in tumors of the adrenal medulla and the ganglia of the sympathetic nervous system. Until that time (October, 1955) there had been 27 such cases recorded in the literature, including Schall and Lineback's, Seaman's, and the original cases of Berger, Luc and Richard. No sex or age predilections were noted. The majority of these tumors, unlike their adrenal or ganglionic prototypes, have been benign. These growths were radiosensitive. Fisher added one case of his own, similar to Schall and Lineback's. In the histological sections, pseudo-rosettes were seen. This tumor was extermely radiosensitive, but the patient ultimately developed pulmonary metastases and died.

The term "neuro-epithelioma" was confusing to this author, as apparently other pathologists mean something else by this term. Eggston and Wolff[6] described one malignant tumor in the nose, which they called neuro-epithelioma, arising from olfactory epithelium. Histologically, it showed a papillary structure, totally unlike our present case. Ewing[7] uses the term neuro-epithelioma to designate a rather well defined group of brain tumors. They consist of cuboidal or cylindrical cells, with neurofibrils, often arranged in palisade form, and presenting so-called rosettes about central spaces, or pseudo-rosettes about blood vessels. They are rapidly growing, infiltrative and malignant. Aside from the brain, they occur in the retina, the abdominal sympathetics, adrenal gland and spinal cord.

This author favors the view that the case reported here is a nonchromaffin paraganglioma, but differences between nonchromaffin paraganglioma, as described herein, and the esthesio-neuro-epithelioma of Berger, Luc and Richards[4] and of Seaman,[19] and the neuroblastoma of Schall and Lineback[18] are so slight as to make one suspect that we are using different terminology for one and the same tumor. The differences in the pathologists' opinions emphasize the difficulty in reaching a diagnosis in certain nasal tumors. Our practical interests were, first, in considering whether or not we were dealing with a malignant lesion; second, what the prog-

nosis was; and third, how would the tumor respond to irradiation. The answer to the first question would seem to be that this tumor was benign; to the second question, it is too early to give an answer; to the third question, it is not likely that we will ever know the answer, as irradiation was not really given a trial until after the tumor had been removed.

CONCLUSION.

The case of a massive tumor of the nasal sinuses and nasal fossae, occurring in a woman in her sixth decade is presented. The tumor was a slow-growing lesion, known to have been present for at least four years. Several pathologists studied the sections of this lesion, and although there were rather marked differences of opinion among them, the evidence seems to favor a diagnosis of nonchromaffin paraganglioma, although it may fall in the category of neuro-epithelioma. The tumor was well-encapsulated, originating by a short pedicle in the posterior ethmoid area. It extended by direct pressure into all the sinuses and the nasal fossae but showed no invasion. After several piecemeal excisions through the nostril the entire tumor was removed via an external approach. This was followed by deep X-ray therapy to the tumor-bearing area. Now after one year the patient is asymtomatic and shows no sign of recurrence.

BIBLIOGRAPHY.

1. Ackerman, L. V. and del Regato, J. A.: "Cancer." Second edition, pp. 205 and 384-385, 1954. C. V. Mosby Co., St. Louis.

2. Anderson, W. A. D.: "Pathology", Second Edition, p. 1175, 1953. C. V. Mosby Co., St. Louis.

3 Andrew, A. H., Jr.: Glomus Tumors (Nonchromaffin Paragangliomas) of the Larynx. *Ann. Otol., Rhinol. and Laryngol.*, 64:1034-1036, Dec., 1955.

4. Berger, Luc, and Richard: l'Esthesio-neuroepitheliome Elfactif. *Bull. d' Assoc. Francais pour l'Etude du Cancer*, 13:410, May, 1924.

5. Bloom, F.: Structure and Histogenesis of Tumors of the Aortic Bodies in Dogs. *Arch. Path.*, 36:1-12, July, 1943.

6. Eggston, A. A., and Wolff, D.: "Histopathology of the Ear, Nose and Throat." First edition, pp. 809,810, 1947. Williams & Wilkins Co., Balt.

7. Ewing, James: "Neoplastic Diseases." Fourth edition, pp. 384, 442, 782, 1942. W. B. Saunders Co., Phila.

8. Fisher, E. R.: Neuroblastoma of the Nasal Fossa. *Archives of Pathology*, 60:435-439, Oct., 1955.

9. Guild, S. R.: A Hitherto Unrecognized Structure, the Glomus Jugularis in Man *Anat. Rev.*, 79; Suppl. No. 2, p. 29, March, 1941.

10. Guild, S. R.: The Glomus Jugulare, a Nonchromaffin Paraganglion, in Man. *Ann. Otol., Rhinol. and Laryngol.*, 62:1045-1071, Dec., 1953.

11. Hollinshead, W. H.: Chromaffin Tissue and Paraganglia. *Quart. Rev. Biol.*, 15:156-171, June, 1940.

12. Lattes, R., and Waltner, J. G.: Nonchromaffin Paraganglioma of the Middle Ear (Carotid-Body-Like Tumor: Glomus Jugulare Tumor). *Cancer*, 2:447-468, May, 1949.

13. Lattes, R.: Nonchromaffin Paraganglioma of Ganglion Nodosum, Carotid Body, and Aortic-Arch Bodies. *Cancer*, 3:667-694, July, 1950.

14. Maximow, A. A., and Bloom, W.: A "Textbook of Histology," pp. 708-709, 1930. W. B. Saunders Co., Phila.

15. Mulligan, R. M.: Chemodectoma in the Dog. (Abstr.) *Am. Jour. Path.*, 26:680-681, July, 1950.

16. Romanski, R.: Chemodectoma (Non-Chromaffima Paraganglioma) of the Carotid Body with Distant Metastases. *Amer. Jour. of Path.*, 30:1-14, Jan.-Feb., 1954.

17. Rosenwasser, K.: Carotid Body Tumor of the Middle Ear and Mastoid. *Arch. Otolaryngol.*, 41:64-67, Jan., 1945.

18. Schall, L. A., and Lineback, M.: Primary Intranasal Neuroblastoma. *Ann. of Otol., Rhinol. and Laryngol.*, 60:221-229, March, 1951.

19. Seaman, W. B.: Olfactory Esthesioneuro-epithelioma. *Radiology*, 57:541-546, Oct., 1951.

20. Ward, G. E., and Hendrick, J. W.: "Tumors of the Head and Neck." First edition, p. 639, 1950. Williams & Wilkins Co., Balt.

21. Williams, H. L., Childs, D. S., Jr., Parkhill, E. M., and Pugh, D. G.: Chemodectomas of the Glomus Jugulare (Nonchromaffin Paraganglioma) with Especial Reference to Their Response to Roentgen Therapy. *Ann. Otol., Rhinol. and Laryngol.*, 64:546-566, June, 1955.

22. Willis, R. A.: "Pathology of Tumors." pp. 715 and 872, 1947. Butterworth & Co., London.

23. Winship, T., Klopp, C. T., and Jenkins, W. H.: Glomus-Jugularis Tumors. *Cancer*, 1:441-448, Sept., 1948.

INTERNATIONAL VOICE CONFERENCE.

Following the International Congress of Otolaryngology in Washington, D. C., next Spring, there will be an International Voice Conference (Laryngeal Research, Function and Therapy) in Chicago, Illinois, May 20-22, 1957. For Information address: Dr. Hans von Leden, 30 North Michigan, Chicago 2, Ill., U.S.A.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The meeting dates of the Sixth International Congress of Otolaryngology are again emphasized as May 5th through May 10th, 1957. The scientific program for the Plenary Sessions is now complete and is as follows:

CHRONIC SUPPURATION OF THE TEMPORAL BONE.

OPENERS: Marcus Diamant, Central County Hospital, Halmstad, Sweden—Anatomical Etiological Factors in Chronic Middle Ear Discharge.

Luzius Rüedi, Zurich, Switzerland—Pathogenesis and Treatment of Cholesteatoma in Chronic Suppuration of the Temporal Bone.

Horst Wullstein, Director, Otolaryngological Clinic, University of Würzburg, Germany—Surgical Repair for Improvement of Hearing in Chronic Otitis Media.

DISCUSSERS: A. Tumarkin, Liverpool, England; Juan Manuel Tato, Buenos Aires, Argentina; T. E. Cawthorne, London, England; Fritz Zöllner, Freiburg, Germany.

COLLAGEN DISORDERS OF THE RESPIRATORY TRACT.

OPENERS: Hans Selye, Director, Institute of Experimental Medicine and Surgery, University of Montreal, Faculty of Medicine, Montreal, Canada.

Introduction:

Michele Arslan, Padua, Italy—The Upper Respiratory Tract.

Leslie Gay, Physician-in-Charge, Allergy Clinic, The Johns Hopkins Hospital, Baltimore, U. S. A.—The Lower Respiratory Tract.

DISCUSSERS: Victor E. Negus, London, England; Branimir Gusic, Zagreb, Yugoslavia; Aubrey G. Rawlins, San Francisco, U. S. A.; Henry L. Williams, Rochester, Minn., U. S. A.

PAPILLOMA OF THE LARYNX.

OPENERS: Jo Ono, Tokyo, Japan—Etiology.

Plinio de Mattos Barretto, Faculty of Medicine, University of Sao Paulo, Brazil.

Diagnosis:

F. C. W. Capps, London, England—Therapy.

DISCUSSERS: C. A. Hamberger, Göteborg, Sweden; Pedro Hernandez Gonzalo, Havana, Cuba; Eelco Huizinga, Groningen, Netherlands; Albert von Riccabona, Vienna, Austria.

Anyone planning to attend the Congress and who has not yet registered should do so immediately in order to obtain hotel registration priority.

For more detailed information pertaining to the Sixth International Congress please communicate with the General Secretary, 700 N. Michigan Ave., Chicago 11, Ill., U. S. A.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

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Any qualified physician may become a Member of the Congress by paying a registration fee of $25.00, U. S. A. Non-medical personnel may register as Associate Members for a fee of $10.00, U. S. A. The registration fee includes the privilege of attending all official meetings of the Congress except the banquet for which an additional charge will be made. Those planning to attend the Congress should complete *Form A* and return it to the General Secretary.

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Annual Meeting: Palmer House, Chicago, Illinois, October, 1957.

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President: Dr. D. M. Lierle, University Hospital, Iowa City, Iowa.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1957.

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

President: Dr. Irvin J. Fine, 506 New Brunswick Ave., Perth Amboy, N. J.
Secretary: Dr. William Schwartz, 224 Lexington Ave., Passaic, N. J.
Meetings: Quarterly.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Joseph A. Sullivan, 174 St. George St., Toronto 5, Canada.
Secretary: Dr. Arthur L. Jones, 611 Brown Bldg., Lexington, Ky.
Treasurer: Dr. Lawrence H. Boies, University Hospital, Minneapolis 14, Minn.
Meeting: Palmer House, Chicago, Ill., October, 1957.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

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Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Arnold L. Caron, 36 Pleasant St., Worchester, Mass.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Jose Gros, Havana, Cuba.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Sixth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Brazil, 1958.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago 11, Ill.
Meeting: Statler Hotel, Washington, D. C., May 5 - 10, 1957.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

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Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. Harry P. Schenck, Dr.; Benjamin H. Shuster, Dr. William A. Lell, Dr.; William J. Hitschler.

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Vice-President: Dr. Frank Wojniak, 6132 So. Kedzie Ave., Chicago, Ill.
Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago, Ill.
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President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
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Vice-Chairman: Dr. Russell Page.
Secretary: Dr. James J. McFarland.
Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Vice-Chairman: Dr. Walter T. Hotchkiss, 541 Lincoln Road, Miami Beach, Florida.
Chairman-Elect: Dr. V. Eugene Holcombe, Medical Arts Building, Charleston, West Virginia.
Secretary: Dr. G. Slaughter Fitz-Hugh, 104 East Market Street, Charlottesville, Virginia.

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President-Elect: Dr. Emanuel U. Wallerstein, Professional Building, Richmond, Virginia.
Vice-President: Dr. Calvin T. Burton, Medical Arts Building, Roanoke, Virginia.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Virginia.
Meeting: Roanoke, Virginia, December 6 and 7, 1957.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.
Annual Meeting: Greenbrier, White Sulphur Springs, W. Va., May 31st through June 1st.

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Vice-President: Dr. A. J. McComiskey, 3420 Prytonia St., New Orleans, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: The Edgewater Gulf Hotel, Edgewater Park, Miss., May 17-18, 1957.

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Meeting:

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

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Vice-President: Dr. Max M. Kulvin.
Secretary-Treasurer: Dr. James H. Mendel, Jr., 7241 Red Road, Miami 43, Florida.
Meeting quarterly (March, May, October and December), on the second Thursday of the month, 6:30 P.M. at Seven Seas Restaurant.

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LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Chairman of Otolaryngology Section: Dr. Howard G. Gottschalk.
Secretary of Otolaryngology Section: Dr. Robert W. Godwin.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P. M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: H. Leroy Goss, M.D., 620 Cobb Bldg., Seattle 1, Washington.
Secretary-Treasurer: Homer E. Smith, M.D., 508 East South Temple, Salt Lake City, Utah.
Meeting: April 7-11, 1957.

THE SOCIETY OF MILITARY OTOLARYNGOLOGISTS.

President: Col. Wendell A. Weller.
Secretary-Treasurer: Major Stanley H. Bear, M.C., 3810th USAF Hospital, Maxwell AFB, Alabama.
Time and place of meeting: October 1957, Palmer House, Chicago, Ill.

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Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.
Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

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President: Dr. G. M. T. Hazen, 208 Canada Bldg., Saskatoon, Sask.
Secretary: Dr. G. Arnold Henry, 170 St. George St., Toronto, Ontario.
Meeting: Banff Springs Hotel, Banff, Canada, June 17-19, 1957.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Theodor Hunermann, Dusseldorf, Germany.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadephia 40, Pa., U. S. A.
Meeting: Sixth International Congress of Bronchoesophagology, Philadelphia, May 12-13, 1957.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting: Lima, Peru, 1957.

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